INVENTOR SEARCH

=> d ibib abs l5 1

L5 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:612039 HCAPLUS Full-text
DOCUMENT NUMBER: 143:112119
TITLE: Process and device for determination of cell viability
INVENTOR(S): Chaiken, Joseph; Dracker, Robert; Hagrman, Pamela J.; Hagrman, Douglas
PATENT ASSIGNEE(S): Lightouch Medical, Inc., USA
SOURCE: PCT Int. Appl., 22 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005063119	A1	20050714	WO 2004-US43759	20041222
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2549014	A1	20050714	CA 2004-2549014	20041222
EP 1699351	A1	20060913	EP 2004-815765	20041222
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS				
US 20070177143	A1	20070802	US 2006-596115	20060531
PRIORITY APPLN. INFO.:			US 2003-531848P	P 20031222
			WO 2004-US43759	W 20041222

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Described is a method to determine the viability of cells by measuring the absolute and relative rate of metabolic activity and/or integrity of the cell membrane through the use of vibrational spectroscopy. The use of deuterated agents facilitates detection of changes associated with a change in viability.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

RESULTS FROM SEARCHES IN CAPLUS, MEDLINE, BIOSIS, EMBASE, DRUGU, AND WPIDS

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=> d que stat l22
L6       822558 SEA FILE=HCAPLUS ABB=ON  (?CELL? OR ?MEMBRAN?)(5A)(?VIABIL? OR
                ?LIVING? OR ?LIVE? OR ?PROLIF? OR ?MULT? OR ?GROW? OR ?DUPLICAT
                ? OR ?METABOL?)
L7         8372 SEA FILE=HCAPLUS ABB=ON  L6 AND (?RAMAN? OR ?INFRARED? OR
                ?INFRA?(W)RED? OR ?NEAR?(W)(?INFRARED? OR ?SPECTROSC? OR
                ?SPECTROSC?))
L8           32 SEA FILE=HCAPLUS ABB=ON  L7 AND (?DEUTERAT? OR ?DEUT? OR
                ?TRIT?)(L)(?MATERIAL? OR ?METABOL? OR ?GLUCOS? OR ?DEXTROS? OR
                ?SUBSTITUENT?)
L9         8372 SEA FILE=HCAPLUS ABB=ON  L7 OR L8
L10           5 SEA FILE=HCAPLUS ABB=ON  L9 AND (?TRITIAT?(4A)(?THIAMEDENE? OR
                ?THYMIDINE?))
L11        8372 SEA FILE=HCAPLUS ABB=ON  L9 OR L10
L12        2723 SEA FILE=HCAPLUS ABB=ON  L11 AND (?BACTER? OR ?MICROB? OR
                ?FUNG? OR ?PROTOZ? OR D20 OR ACID? OR ?WATER? OR ?DEUTERIUM?)
L13        2723 SEA FILE=HCAPLUS ABB=ON  L12 AND (?RAMAN? OR ?INFRARED? OR
                ?INFRA?(W)RED? OR ?NEAR?(W)(?INFRARED? OR ?SPECTROSC? OR
                ?SPECTROSC?))
L14          22 SEA FILE=HCAPLUS ABB=ON  L13 AND (?DEUTERAT? OR ?DEUT? OR
                ?TRIT?)(L)(?MATERIAL? OR ?METABOL? OR ?GLUCOS? OR ?DEXTROS? OR
                ?SUBSTITUENT?)
L15           4 SEA FILE=HCAPLUS ABB=ON  L14 AND ?INTEG?
L16          22 SEA FILE=HCAPLUS ABB=ON  L14 OR L15
L17          34 SEA L16
L18          40 DUP REMOV L16 L17 (16 DUPLICATES REMOVED)
L19          27 SEA FILE=WPIDS ABB=ON  L14 OR L15
L20          66 DUP REMOV L18 L19 (1 DUPLICATE REMOVED)
L21          28 SEA L20 AND (PRD<20031222 OR PD<20031222)
L22          66 SEA L20 OR L21


=> d ibib abs l22 1-66

L22  ANSWER 1 OF 66  HCAPLUS  COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER:         2009:1156250  HCAPLUS  Full-text
DOCUMENT NUMBER:          151:398024
TITLE:                    In Situ Metabolic Profiling of Single
                          Cells by Laser Ablation Electrospray
                          Ionization Mass Spectrometry
AUTHOR(S):                Shrestha, Bindesh; Vertes, Akos
CORPORATE SOURCE:         Department of Chemistry, W. M. Keck Institute for
                          Proteomics Technology and Applications, The George
                          Washington University, Washington District of
                          Columbia, 20052, USA
SOURCE:                   Analytical Chemistry (Washington, DC, United States)
                          (2009), 81(20), 8265-8271
                          CODEN: ANCHAM; ISSN: 0003-2700
PUBLISHER:                American Chemical Society
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB    Depending on age, phase in the cell cycle, nutrition, and environmental
      factors, individual cells exhibit large metabolic diversity.  To explore
      metabolic variations in cell populations, laser ablation electrospray
      ionization (LAESI) mass spectrometry (MS) was used for the in situ anal. of
      individual cells at atmospheric pressure.  Single cell ablation was achieved
      by delivering mid-IR laser pulses through the etched tip of a GeO2-based glass
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fiber. Metabolic anal. was performed from single cells and small cell populations of Allium cepa and Narcissus pseudonarcissus bulb epidermis, as well as single eggs of Lytechinus pictus. Of the 332 peaks detected for A. cepa, 35 were assigned to metabolites with the help of accurate ion masses and tandem MS. The metabolic profiles from single cells of the two plant species included a large variety of oligosaccharides including possibly fructans in A. cepa, and alkaloids, e.g., lycorine in N. pseudonarcissus. Anal. of adjacent individual cells with a difference in pigmentation showed that, in addition to essential metabolites found in both variants, the pigmented cells contained anthocyanidins, other flavonoids, and their glucosides. Anal. of single epidermal cells from different scale leaves in an A. cepa bulb showed metabolic differences corresponding to their age. The authors' results indicate the feasibility of using LAESI-MS for the in situ anal. of metabolites in single cells with potential applications in studying cell differentiation, changes due to disease states, and response to xenobiotics.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 2 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2009:924080 HCAPLUS Full-text

DOCUMENT NUMBER: 151:344531

TITLE: Nitric oxide-releasing S-nitrosothiol-modified xerogels

AUTHOR(S): Riccio, Daniel A.; Dobmeier, Kevin P.; Hetrick, Evan M.; Privett, Benjamin J.; Paul, Heather S.; Schoenfisch, Mark H.

CORPORATE SOURCE: Department of Chemistry, Caudill Laboratories, University of North Carolina, Chapel Hill, NC, 27599, USA

SOURCE: Biomaterials (2009), 30(27), 4494-4502 CODEN: BIMADU; ISSN: 0142-9612

PUBLISHER: Elsevier Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The synthesis, material characterization, and in vitro biocompatibility of S-nitrosothiol (RSNO)-modified xerogels are described. Thiol-functionalized xerogel films were formed by hydrolysis and co-condensation of 3-mercaptopropyltrimethoxysilane (MPTMS) and methyltrimethoxysilane (MTMOS) sol-gel precursors at varying concns. Subsequent thiol nitrosation via acidified nitrite produced RSNO-modified xerogels capable of generating nitric oxide (NO) for up to 2 wk under physiol. conditions. Xerogels also exhibited NO generation upon irradiation with broad-spectrum light or exposure to copper, with NO fluxes proportional to wattage and concentration, resp. Xerogels were capable of storing up to .apprx.1.31 μmol NO mg-1, and displayed negligible fragmentation over a 2-wk period. Platelet and bacterial adhesion to nitrosated films was reduced compared to non-nitrosated controls, confirming the antithrombotic and antibacterial properties of the NO-releasing materials. Fibroblast cell viability was maintained on the xerogel surfaces illustrating the promise of RSNO-modified xerogels as biomedical device coatings.

REFERENCE COUNT: 63 THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 3 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2009:854992 HCAPLUS Full-text

TITLE: Chemical, biotechnological and pharmacological application of tomato hybrids

AUTHOR(S): Tommonaro, G.; Poli, A.; De Prisco, R.; Nicolaus, B.

CORPORATE SOURCE: Institute of Biomolecular Chemistry, Pozzuoli (Na), 80078, Italy

SOURCE: Acta Horticulturae (2009), 823(Proceedings of the XIth

International Symposium on the Processing Tomato, 2008), 49-58
CODEN: AHORA2; ISSN: 0567-7572
PUBLISHER: International Society for Horticultural Science
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In collaboration with M.F.M. International of Torre del Greco we studied the nutritional quality of several tomato hybrids of Solatium lycopersictim L. Samples were collected from field trials conducted at several sites with different pedoclimatic conditions. The hydrophilic, lipophilic and methanolic antioxidative activities of these hybrids exts. were determined The hydrophilic and lipophilic antioxidant activity was carried out by DMPD (N,N-Dimethyl-p-phenylenediamine dihydrochloride) and ABTS (2,2'-Azino-bis(3-ethylbenzothiazoline-6-sulfonic acid) diammonium salt) methods, resp. The acetonic antioxidant activity was performed by the DPPH method and, on this fraction, total polyphenolic content was evaluated by Folin-Ciocalteau assay. To evaluate potential anti-tumoral activity, cytotoxic activities by brine shrimps assay were also determined in all acetonic exts. Moreover the purification of the polysaccharide fraction derived from products and byproducts of the tomato industry (Solanum lycopersicum L.) and its chemical composition, rheol. properties and partial primary structure on the basis of spectroscopic analyses (UV, IR, 1H NMR) will be reported. We have investigated the effect of the polysaccharide on nitrite and ROS production in LPS-stimulated J774 macrophages for 24 h. The tomato peel polysaccharide was inhibited in a concentration-dependent nitrite and ROS production as well as iNOS protein expression induced by LPS. Incubation of cells with the polysaccharide resulted in a significant decrease of NF-kB/DNA binding activity which was correlated with a marked reduction of iNOS mRNA levels. These results show that polysaccharide inhibits NF-kB activation and iNOS gene expression by preventing the reactive species production, and suggest for this compound a role for controlling oxidative stress and/or inflammation. The achievements of biodegradable and thermoplastic materials by using chemical and enzymic processes from polysaccharides are also reported. The realization of three and more manufactured articles, for example flowerpots or rigid packing vases, film for agriculture use and ensiling film will be in progress.
REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 4 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2009:754391 HCAPLUS Full-text
TITLE: Age-associated impairment of Akt phosphorylation in primary rat hepatocytes is remediated by alpha-lipoic acid through PI3 kinase, PTEN, and PP2A
AUTHOR(S): Petersen Shay, Kate; Hagen, Tory M.
CORPORATE SOURCE: Linus Pauling Institute, Oregon State University, Corvallis, OR, 97331-6512, USA
SOURCE: Biogerontology (2009), 10(4), 443-456
CODEN: BIOGCN; ISSN: 1389-5729
PUBLISHER: Springer
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Akt is a highly regulated serine/threonine kinase involved in stress response and cell survival. Stress response pathways must cope with increasing chronic stress susceptibility with age. We found an age-related lesion in Akt activity via loss of phosphorylation on Ser473. In hepatocytes from old rats, basal phospho-Ser473 Akt is 30% lower when compared to young, but basal phospho-Thr308 Akt is unchanged. (R)-α-lipoic acid (LA), a dithiol compound with antioxidant properties, is effective against age-related increases in oxidative stress and has been used to improve glucose utilization through insulin receptor (IR) pathway-mediated Akt phosphorylation. Treatment with

physiol. relevant doses of LA (50 μM) provided a 30% increase in phospho-Ser473. Furthermore, two phosphatases that antagonize Akt, PTEN and PP2A, were both partially inhibited by LA. Thus, LA may be a nutritive agent that can remediate loss of function in the Akt pathway and aid in the survival of liver cells.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 5 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2008:1493824 HCAPLUS Full-text
DOCUMENT NUMBER: 151:168005
TITLE: Effects of fungus inoculation and salt stress on physiology and biochemistry of in vitro grapevines: Emphasis on sugar composition changes by FT-IR analyses
AUTHOR(S): Oliveira, Helena; Barros, Antonio S.; Delgadillo, Ivonne; Coimbra, Manuel A.; Santos, Conceicao
CORPORATE SOURCE: Department of Biology, University of Aveiro, Aveiro, 3810-193, Port.
SOURCE: Environmental and Experimental Botany (2009), 65(1), 1-10
CODEN: EEBODM; ISSN: 0098-8472
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The impacts of salt stress and inoculation in in vitro grapevine (Vitis vinifera L.) growth, nutrient accumulation, osmoregulation, photosynthesis and membrane integrity were evaluated. One month exposure to 100 mM NaCl as well as to inoculation with Phaeomoniella chlamydospora reduced relative growth rate (RGR) and induced senescence in grapevine plants, shown by: (1) decrease of Ψ π without osmoregulation, (2) decrease of chlorophyll content and fluorescence, (3) loss of membrane integrity and (4) nutritional disorders. To assess putative changes in structural and/or non-structural carbohydrates induced by these two stress conditions, alc. insol. residues from the roots, stems and leaves were also characterized by FT-IR and GC with respect to the sugar composition The referred organs were distinguished based on: (1) higher proportion of uronic acid residues in leaves which diagnose the presence of pectic polysaccharides (wavenumbers 1100, 1150 and 1018 cm-1 in FT-IR spectra), (2) higher proportion of xylose and glucose on stems and FT-IR spectra diagnostic of xylose-rich polysaccharides (1041 cm-1) and cellulose (1060 cm-1), (3) higher proportion of glucose residues, xylose and arabinose on roots and a FT-IR spectra characteristic of xylose-rich polysaccharides (1041 cm-1). The main alterations induced by salt stress and inoculation were more visible in leaves, where the content of uronic acid decreased showing that changes in cell wall composition occurred, mostly at the pectic fraction. Besides, an accumulation of insol. glucose was found, and FT-IR spectra showed that this glucose-based material was starch (maximum absorption at 998 cm-1), accumulated as a non-specific response to salt stress and P. chlamydospora inoculation.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)
REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 6 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2008:1240727 HCAPLUS Full-text
DOCUMENT NUMBER: 150:128840
TITLE: Osteoblast interaction with DLC-coated Si substrates
AUTHOR(S): Chai, Feng; Mathis, Nicolas; Blanchemain, Nicolas; Meunier, Cathy; Hildebrand, Hartmut F.
CORPORATE SOURCE: Groupe de Recherche sur les Biomateriaux (GRB),

Laboratoire de Biophysique, UPRES EA 1049, Faculte de Medecine, Universite de Lille-2, Lille, 59045, Fr.
SOURCE: Acta Biomaterialia (2008), 4(5), 1369-1381
CODEN: ABCICB; ISSN: 1742-7061
PUBLISHER: Elsevier Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Diamond-like carbon (DLC) coating is a convenient means of modifying material surfaces that are sensitive to wear, such as titanium and silica substrates. This work aims to evaluate the osteoblast-like cells' response to DLC-coated Si (Si-DLC), which was treated under different conditions. DLC and deuterated DLC films were deposited by plasma-enhanced chemical vapor deposition to obtain a 200-nm-thick layer on all the samples. Three types of precursor gas were applied for deposition: pure methane (CH4), pure deuterated methane (CD4) and their half/half mixture All surface treatments were performed under two different self-bias voltages (Vsb): -400 and -600 V. The modified surfaces were characterized by XPS, Raman spectroscopy, Rutherford backscattering spectroscopy, elastic recoil detection anal., X-ray reflectometry and the sessile-drop method. MC3T3-E1 osteoblasts were cultured on the Si-DLC wafers for 3 and 6 days. Biol. tests to measure cell proliferation, cell vitality, cell morphol. and cell adhesion were performed. All DLC coatings produced a slightly more hydrophobic state than non-treated Si. Certain types of amorphous DLC coating, such as the surface treated under the Vsb of -600 V in pure methane (600CH4) or in pure deuterated methane (600CD4), offered a significantly higher cell proliferation rate to Si substrate. SEM observations confirmed that the optimal cell adhesion behavior, among all the treated surfaces, occurred on the surface of the 600CH4 and 600CD4 groups, which showed increased amts. of filopodia and microvilli to enhance cell-environment exchange. In conclusion, DLC coating on Si could produce better surface stability and improved cellular responses. OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)
REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 7 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2008:880222 HCAPLUS Full-text
DOCUMENT NUMBER: 151:300663
TITLE: Influence of lithium ions on the NLO properties of KDP single crystals
AUTHOR(S): Shirsat, M. D.; Hussaini, S. S.; Dhumane, N. R.; Dongre, V. G.
CORPORATE SOURCE: Optoelectronics and Sensor Research Laboratory, Department of Physics, Dr. Babasaheb Ambedkar Marathwada University, Aurangabad, 431004, India
SOURCE: Crystal Research and Technology (2008), 43(7), 756-761
CODEN: CRTEDF; ISSN: 0232-1300
PUBLISHER: Wiley-VCH Verlag GmbH & Co. KGaA
DOCUMENT TYPE: Journal
LANGUAGE: English

AB K Dihydrogen Phosphate (KDP) and its isomorphous deuterated form are popular due to their applications in frequency converters and electrooptic modulation. Different attempts were made to dope KDP with inorg. additives, organic materials and amino acids. Since many of the metal ions possess more electro negativity which increases the non centrosymmetry, it is of interest to dope them in KDP. The influence of Li ion (Li+) on NLO properties of KDP crystal was studied. Single crystal of Li ion doped K Dihydrogen Phosphate (KDP) was grown by slow evaporation technique. The enhancement in SHG efficiency after addition of ion Li (Li+) was observed by Kurtz Powder SHG test. The SHG efficiency of KDP after addition of Li ion is 1.33 times more than pure KDP. The crystal structure and cell parameters of grown crystal were determined by x-ray diffraction. The Energy Dispersive x-ray

anal. (EDAX) gives the chemical composition of grown crystal. The functional groups were identified by FTIR spectral anal. The presence of Li in the material of grown crystal was detected by Atomic absorption spectroscopy (AAS). The optical absorption and transmission studies were done by UV-Visible spectral anal. The grown crystal was subjected to TGA and Differential Scanning Calorimetry (DSC).
OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 8 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2007:700356 HCAPLUS Full-text
DOCUMENT NUMBER: 147:482839
TITLE: The environmental plasmid pQBR103 alters the single-cell Raman spectral profile of Pseudomonas fluorescens SBW25
AUTHOR(S): Ude, Susanne; Bailey, Mark J.; Huang, Wei E.; Spiers, Andrew J.
CORPORATE SOURCE: Department of Plant Sciences, University of Oxford, Oxford, OX1 3RB, UK
SOURCE: Microbial Ecology (2007), 53(3), 494-497
CODEN: MCBEBU; ISSN: 0095-3628
PUBLISHER: Springer
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Although plasmids are ubiquitous amongst phytosphere pseudomonads, the advantage and costs of plasmids for the bacterial host remain unclear. The application of single-cell Raman spectral anal. to plasmid-bacterial systems under different environmental conditions offers a new means of determining the impact of plasmids on host cell physiol., metabolic status, and response to stress. OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 9 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2007:531562 HCAPLUS Full-text
DOCUMENT NUMBER: 147:7458
TITLE: Clinical significance of glucose metabolism in type C chronic liver disease
AUTHOR(S): Oinuma, Goro
CORPORATE SOURCE: Div. Gastroenterol. Hepatol., Dep. Med., Sch. Med., Nihon University, Japan
SOURCE: Nichidai Igaku Zasshi (2007), 66(2), 173-178
CODEN: NICHAS; ISSN: 0029-0424
PUBLISHER: Nihon Daigaku Igakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB It was recently reported that the manifestation and mortality of hepatocellular carcinoma increase in cases with chronic type C hepatitis and the co-occurrence of diabetes and obesity. This suggests the value of interventional nutrition therapy for cancer prevention in type C chronic liver disease. In the present study, we performed a 75 g oral glucose tolerance test (OGTT) in type C chronic liver disease patients to determine the status of glucose metabolism and its clin. significance. Glucose intolerance was observed in 45.7% of chronic hepatitis and 76.6% of liver cirrhosis cases. Homeostasis model assessment for insulin resistance (HOMA-IR) showed insulin resistance in 20.5% of chronic hepatitis and 47.7% of liver cirrhosis cases. The plasma glucose daily profile showed a maximum of 200 mg/dL or higher in 27.3% of glucose intolerance cases and 66.7% of insulin resistance cases by a

75 g OGTT. Our findings suggest that a 75 g OGTT should actively be carried out in chronic type C liver disease patients and that interventional nutrition therapy should be given in borderline and diabetes-type glucose intolerance cases.

L22 ANSWER 10 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2007:332144 HCAPLUS Full-text
DOCUMENT NUMBER: 148:308003
TITLE: Synthesis and plant growth-regulating activity of ethyl α-[[(benzylidene)amino]phenoxy]propionate
AUTHOR(S): Liu, Guo-Hua; Yu, Han; Yao, Mei; Fang, Hai-Bin
CORPORATE SOURCE: Department of Chemistry, College of Life and Environment Science, Shanghai Normal University, Shanghai, 200234, Peop. Rep. China
SOURCE: Yingyong Huaxue (2007), 24(2), 152-156
CODEN: YIHUED; ISSN: 1000-0518
PUBLISHER: Kexue Chubanshe
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
OTHER SOURCE(S): CASREACT 148:308003

AB Eleven Et α-[[(benzylidene)amino]phenoxy]propionate derivs. were synthesized through coupling of aromatic aldehydes with Et α-(4-aminophenoxy)propionate. All compds. were characterized by means of 1H NMR, UV, IR and elemental anal. The effects of substituents of aromatic aldehyde on reaction yields were studied. The results showed that the coupling reaction using 4-nitrobenzaldehyde with electron-withdrawing substituents gave the highest yield in 93%, while the lowest reaction yield of 54% was obtained using 4-methylbenzaldehye, which has electron-donating substituents. The preliminary biol. tests show that most of the compds. possess plant growth-regulating activities at a low concentration (10 ppm). Among those compds., two compds. show root growth-regulating activities of 52% and 55% for Cucumis sativa L., resp.; while another compound gives a inhibitory activity of 75% for root of Cucumis Sativa L. 2-[4-[[(3-Nitrophenyl)methylene]amino]phenoxy]propanoic acid Et ester also gives a cell-growing activity of 51% for Triticum aestivum L. (wheat) and 2-[4-[[(4-methylphenyl)methylene]amino]phenoxy]propanoic acid Et ester gives a stalk growth-regulating activity of 53% for Cucumis Sativa L.

L22 ANSWER 11 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2006:979567 HCAPLUS Full-text
DOCUMENT NUMBER: 146:311918
TITLE: Spectroscopic properties of chalcogenide fibres for biosensor applications
AUTHOR(S): Lucas, Pierre; Solis, Michelle. A.; Juncker, Christophe; Le Coq, David; Riley, Mark R.; Collier, Jayne; Boesewetter, Dianne E.; Boussard-Pledel, Catherine; Bureau, Bruno
CORPORATE SOURCE: Department of Material Science and Engineering, University of Arizona, Tucson, AZ, 85721, USA
SOURCE: Physics and Chemistry of Glasses: European Journal of Glass Science and Technology, Part B (2006), 47(2), 88-91
CODEN: PCGECL
PUBLISHER: Society of Glass Technology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The spectroscopic characteristics of Te2As3Se5 IR optical fibers are

investigated. Fibers with a diameter of approx. 400 μm are tapered to produce a sensitive sensing zone and used as both a sensor and transmission line of the IR optical signal in evanescent wave spectroscopy expts. The fiber surface is shown to be hydrophobic, which results in enhanced detection sensitivity for nonpolar organic species in aqueous media. The peak intensity of organic species increases systematically relative to the peak intensity of water during a comparison of fiber and transmission spectroscopy expts. A bio-optical sensor is developed by coating the fiber with human lung cells at the surface of the sensing zone. The metabolic activity of the cell is monitored spectroscopically and it is shown that the evanescent wave can locally probe the cell membrane integrity. During exposure to Triton X-100, the cell membrane signal shows a sharp decay in response to the surfactant. The ratio of Me and methylene vibrations from membrane lipids decreases rapidly and provides a sensitive probe of the cell membrane integrity. This experiment demonstrates the capability of these fiber based bio-optic sensors to detect micromolar amts. of environmental toxicant.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 12 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2006:918226 HCAPLUS Full-text

DOCUMENT NUMBER: 145:321633

TITLE: Silicon-coated near-infrared magnetic nanoparticles for tumor treatment

INVENTOR(S): Chu, Maoquan

PATENT ASSIGNEE(S): Tongji University, Peop. Rep. China

SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 20pp. CODEN: CNXXEV

DOCUMENT TYPE: Patent

LANGUAGE: Chinese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1698582	A	20051123	CN 2005-10025587	20050429
PRIORITY APPLN. INFO.:			CN 2005-10025587	20050429

AB The invention provides a silicon-coated near-IR fluorescent magnetic nanoparticle for tumor treatment, which is prepared by embedding magnetic nanoparticle and near-IR fluorescent quantum dot nanoparticle or near-IR fluorescent dye mol. in silicon dioxide particle to form a silicon-coated composite nanoparticle, and binding the silicon-coated composite nanoparticle with antitumor drug and biomol. with tumor-targeting effect (such as antibody, ligand, polypeptide and cytokine). The invention is characterized in the integration of magnetic property, quantum size effect, thermal effect of light, efficacy of antitumor drug, and biomol. recognition; and is a promising approach to tumor treatment. For example, nanoparticles of CdTe and Fe3O4, and adriamycin were mixed in PEG water solution together with Triton X100 and n-hexanol to silica microemulsion to obtain the final antitumor emulsion.

L22 ANSWER 13 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2005:612039 HCAPLUS Full-text

DOCUMENT NUMBER: 143:112119

TITLE: Process and device for determination of cell viability

INVENTOR(S): Chaiken, Joseph; Dracker, Robert; Hagrman, Pamela J.; Hagrman, Douglas

PATENT ASSIGNEE(S): Lightouch Medical, Inc., USA

SOURCE: PCT Int. Appl., 22 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005063119	A1	20050714	WO 2004-US43759	20041222
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2549014	A1	20050714	CA 2004-2549014	20041222
EP 1699351	A1	20060913	EP 2004-815765	20041222
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS				
US 20070177143	A1	20070802	US 2006-596115	20060531
PRIORITY APPLN. INFO.:			US 2003-531848P P	20031222
			WO 2004-US43759 W	20041222

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Described is a method to determine the viability of cells by measuring the absolute and relative rate of metabolic activity and/or integrity of the cell membrane through the use of vibrational spectroscopy. The use of deuterated agents facilitates detection of changes associated with a change in viability. OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 14 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:325295 HCAPLUS Full-text
TITLE: Spectroscopic characterization of microorganisms by Fourier transform infrared microspectroscopy
AUTHOR(S): Yu, Chenxu; Irudayaraj, Joseph
CORPORATE SOURCE: Department of Agricultural and Biological Engineering, Pennsylvania State University, State College, PA, 16802, USA
SOURCE: Biopolymers (2005), 77(6), 368-377
CODEN: BIPMAA; ISSN: 0006-3525
PUBLISHER: John Wiley & Sons, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Spectroscopic fingerprints of bacteria were investigated by Fourier transform IR (FTIR) microspectroscopy for the elucidation of chemical composition and structural information during growth. Good differentiation of six microorganisms was achieved down to the strain level. The inherent compositional and structural differences of cell envelopes and cytoplasm were investigated and utilized to obtain more detailed anal. of the spectroscopic features. Bands or regions of key functional groups were also identified in the original spectra. Microspectroscopic monitoring of bacterial growth demonstrated that FTIR spectroscopy cannot only provide mol. fingerprints of the cell envelope, but also compositional and

metabolic information of the cytoplasm under different physiol. conditions. This approach could be an effective alternative to traditional nutritional and biochem. methods to monitor and assess the effects of inhibitors and other environmental factors on microbial cell growth.
OS.CITING REF COUNT: 26 THERE ARE 26 CAPLUS RECORDS THAT CITE THIS RECORD (27 CITINGS)
REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 15 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2003:865663 HCAPLUS Full-text
DOCUMENT NUMBER: 140:124341
TITLE: Interaction of natural polyamines and dimethylsilyl analogues with a phospholipid monolayer: A study by Brewster angle microscopy and PM-IRRAS
AUTHOR(S): Berdycheva, Olga; Desbat, Bernard; Vaultier, Michel; Saint-Pierre-Chazalet, Michele
CORPORATE SOURCE: Laboratoire de Physicochimie Biomoleculaire et Cellulaire, UMR CNRS 7033, Universite Pierre et Marie Curie, Paris, F-75252/05, Fr.
SOURCE: Chemistry and Physics of Lipids (2003), 125(1), 1-11
CODEN: CPLIA4; ISSN: 0009-3084
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB This work presents an anal. of the physicochem. interactions of natural and dimethylsilyl polyamines with an anionic deuterated phospholipid monolayer, d62DPPG (dipalmitoyl phosphatidyl glycerol), at the air-water interface. It was motivated by previous studies, which suggested an antitumor strategy based on the accumulation of derivs. such as bis(7-amino-4-azaheptyl) dimethylsilyl (azhepSi), in order to diminish the concentration of natural polyamines (spermine and putrescine) whose metabolism is strongly activated in tumor cells. Our results, obtained by the surface-pressure technique, Brewster angle microscopy (BAM) and polarization modulation IR reflection absorption spectroscopy (PM-IRRAS), support the idea of an interaction between the polar head groups of d62DPPG and amino groups followed by an adsorption of polyamines up to the carbonyl group. Moreover, an insertion of the dimethylsilyl group up to the alkyl chains occurs with azhepSi, in agreement with the observation that the cohesion of the alkyl chain is lower in this case, as compared with the effect of natural polyamines. OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 16 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2002:963388 HCAPLUS Full-text
DOCUMENT NUMBER: 139:17105
TITLE: Nitrolinoleate, a nitric oxide-derived mediator of cell function: synthesis, characterization, and vasomotor activity
AUTHOR(S): Lim, Dong Gun; Sweeney, Scott; Bloodsworth, Allison; White, C. Roger; Chumley, Phillip H.; Krishna, N. Rama; Schopfer, Francisco; O'Donnell, Valerie B.; Eiserich, Jason P.; Freeman, Bruce A.
CORPORATE SOURCE: Department of Anesthesiology, University of Alabama at Birmingham, Birmingham, AL, 35233, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2002), 99(25), 15941-15946

CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 139:17105

AB Nitric oxide (*NO) and *NO-derived reactive species rapidly react with lipids during both autocatalytic and enzymic oxidation reactions to yield nitrated derivs. that serve as cell signaling mols. Herein we report the synthesis, purification, characterization, and bioactivity of nitrolinoleate (LNO2). Nitroselenylation of linoleic acid yielded LNO2 that was purified by solvent extraction, silicic acid chromatog., and reverse-phase HPLC. Structural characterization was performed by IR spectroscopy, 15N-NMR, LC-neg. ion electrospray mass spectroscopy (MS), and chemiluminescent nitrogen anal. Quant. MS anal. of cell and vessel LNO2 metabolism, using L[15N]O2 as an internal standard, revealed that LNO2 is rapidly metabolized by rat aortic smooth muscle (RASM) monolayers and rat thoracic aorta, resulting in nitrite production and up to 3-fold increases in cGMP (ED50 = 30 μM for RASM, 50 μM for aorta). LNO2 induced endothelium-independent relaxation of preconstricted rat aortic rings, which was unaffected by LG-nitro-L-arginine Me ester addition and inhibited by the guanylate cyclase inhibitor 1H-[1,2,4] oxadiazole[4,3-a]quinoxalin-1-one and the *NO scavenger HbO2. These results reveal that synthetic LNO2, identical to lipid derivs. produced biol. by the reaction of *NO and *NO-derived species with oxidizing unsatd. fatty acids (e.g., linoleate), can transduce vascular signaling actions of *NO. OS.CITING REF COUNT: 52 THERE ARE 52 CAPLUS RECORDS THAT CITE THIS RECORD (52 CITINGS)
REFERENCE COUNT: 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 17 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2002:528062 HCAPLUS Full-text
DOCUMENT NUMBER: 137:160912
TITLE: Mid-infrared spectroscopy of molecular species that drive significant atmospheric processes
AUTHOR(S): Misra, Prabhakar; Dowdye, Edward H., Jr.
CORPORATE SOURCE: Laser Spectroscopy Laboratory, Department of Physics & Astronomy and Center for the Study of Terrestrial and Extraterrestrial Atmospheres, Howard University, Washington, DC, 20059, USA
SOURCE: Proceedings of the International Conference on Lasers (2002), Volume Date 2001, 24th, 386-393
CODEN: PICLDV; ISSN: 0190-4132
PUBLISHER: STS Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Small changes in the IR absorbance observed at levels of concentration in the sub-ppm (sub-ppm) of atmospherically significant mol. species, such as HCl, SO2, NO2 and NH3, were quantified as a function of pressure and temperature for the surface reactions of these gases with various materials (e.g. Cu, Al, stainless steel and teflon). A Nicolet Magna-IR 550 FTIR spectrometer operating in the 400-4000 cm-1 region and fitted with a 10-m multipass absorption cell and a deuterated triglycine sulfate (DTGS) detector was used for recording the spectra. Tubing materials chosen are typically associated with the hardware of measuring instruments used to collect atmospheric trace gas samples. The degree of adsorption for specific gas-solid interfaces and the kinetic theory of the adsorption process (assuming a mono-layer) enabled the determination of the corresponding rate consts. It is envisioned that the accurate determination of the adsorption coverage parameters and the Langmuir rate consts. for the various gas-material combinations will aid in the refinement of precise values for the associated residence time and activation

energies and thereby lead to the development of a comprehensive adsorption isotherm model for gas-surface interactions.

REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 18 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1998:400207 HCAPLUS Full-text
DOCUMENT NUMBER: 129:81146
ORIGINAL REFERENCE NO.: 129:16765a
TITLE: Dietary docosahexaenoic acid and immunocompetence in young healthy men
AUTHOR(S): Kelley, D. S.; Taylor, P. C.; Nelson, G. J.; Mackey, B. E.
CORPORATE SOURCE: USDA, ARS, Western Human Nutrition Research Center, Presidio of San Francisco, CA, 94129, USA
SOURCE: Lipids (1998), 33(6), 559-566
CODEN: LPDSAP; ISSN: 0024-4201
PUBLISHER: AOCS Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The purpose of this study was to examine the effect of dietary docosahexaenoic acid (DHA), in the absence of eicosapentaenoic acid, on human immune response (IR). A 120-d study with 11 healthy men was conducted at the Metabolic Research Unit of the Western Human Nutrition Research Center. Four subjects (control group) were fed the stabilization or basal diet (15, 30, and 55% energy from protein, fat, and carbohydrate, resp.) throughout the study; the remaining seven subjects (DHA group) were fed the basal diet for the first 30 d, followed by 6 g DHA/d for the next 90 d. DHA replaced an equivalent amount of linoleic acid; the two diets were comparable in their total fat and all other nutrients. Both diets were supplemented with 20 mg d-α-tocopherol acetate per day. Indexes of IR were examined on study day 22, 30, 78, 85, 106, and 113. Addition of DHA at moderately high levels did not alter the proliferation of peripheral blood mononuclear cells cultured with phytohemagglutinin or Con A, or the delayed hypersensitivity skin response. Also, addnl. DHA did not alter the number of T cells producing interleukin 2 (IL2), the ratio between the helper/suppressor T cells in circulation, or the serum concns. of IgG, C3, and interleukin 2 receptor (IL2R). DHA supplementation, however, caused a significant (P = 0.0001) decrease in the number of circulating white blood cells which was mainly due to a decrease in the number of circulating granulocytes. The number of lymphocytes in peripheral circulation was not affected by Dietary DHA enrichment, but the percentage of lymphocytes in white blood cells increased because of a reduction in granulocyte nos. None of these indexes was changed in the control group. Our results show that when total fat intake is low and held constant, DHA consumption does not inhibit many of the lymphocyte functions which have been reported to be inhibited by fish oil consumption.

OS.CITING REF COUNT: 61 THERE ARE 61 CAPLUS RECORDS THAT CITE THIS RECORD (61 CITINGS)
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 19 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1998:176730 HCAPLUS Full-text
DOCUMENT NUMBER: 128:305629
ORIGINAL REFERENCE NO.: 128:60501a,60504a
TITLE: Structure of yeast plasma membrane H+-ATPase: comparison of activated and basal-level enzyme forms
AUTHOR(S): Tanfani, Fabio; Lapathitis, Georgios; Bertoli, Enrico; Kotyk, Arnost
CORPORATE SOURCE: Medical School, Inst. of Biochemistry, Univ. of Ancona, Ancona, 60131, Italy

SOURCE: Biochimica et Biophysica Acta, Biomembranes (1998), 1369(1), 109-118
CODEN: BBBMBS; ISSN: 0005-2736
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Plasma membrane H+-ATPase of the yeast Saccharomyces cerevisiae was isolated and purified in its two forms, the activated A-ATPase from glucose-metabolizing cells, and the basal-level B-ATPase from cells with endogenous metabolism only. Structure of the two enzyme forms and the effects of β,γ-imidoadenosine 5'-triphosphate (AMP-PNP) and of diethylstilbestrol (DES) thereon were analyzed by FT-IR spectroscopy. IR spectra revealed the presence of two populations of α-helixes with different exposure to the solvent in both the A-ATPase and the B-ATPase. AMP-PNP did not affect the secondary structure of A-ATPase while DES affected the ratio of the two α-helix populations. Thermal denaturation expts. suggested a more stable structure in the B-form that in the A-form. AMP-PNP stabilized the A-ATPase structure while DES destabilized both enzyme forms. IR spectra showed that 60% of the amide hydrogens were exchanged for deuterium in both forms at 20°. The remaining 40% were exchanged at higher temps. The maximum amount of H/D exchange was observed at 50-55° for both enzyme forms, while in the presence of DES it was observed at lower temps. The data do not contradict the possibility that the activation of H+-ATPase is due to the C-terminus of the enzyme dissociating from the ATP-binding site which is covered by it in the less active form. OS.CITING REF COUNT: 12 THERE ARE 12 CAPLUS RECORDS THAT CITE THIS RECORD (12 CITINGS)
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 20 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1993:143141 HCAPLUS Full-text
DOCUMENT NUMBER: 118:143141
ORIGINAL REFERENCE NO.: 118:24523a,24526a
TITLE: Sarcina ventriculi synthesizes very long chain dicarboxylic acids in response to different forms of environmental stress
AUTHOR(S): Jung, Seunho; Lowe, Susan E.; Hollingsworth, Rawle I.; Zeikus, J. Gregory
CORPORATE SOURCE: Dep. Biochem., Michigan State Univ., East Lansing, MI, 48824, USA
SOURCE: Journal of Biological Chemistry (1993), 268(4), 2828-35
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Changes in the composition of membrane lipids in a strictly anaerobic, facultative acidophilic eubacterium, Sarcina ventriculi, were studied in response to various forms of environmental stress. Changes in lipid composition and structure occurred in response to changes in environmental pH. At neutral pH, the predominant membrane fatty acids ranged in chain length from C14 to C18. However, when cells were grown at pH 3.0, a family of unique very long chain fatty acids containing 32-36 carbon atoms was synthesized and accounted for 50% of the total membrane fatty acids. These acids were identified as very long chain α,ω-dicarboxylic acids ranging in length from 28 to 36 carbons by electron impact mass spectrometry of Me and (perdeuterio) Me ester derivs. These Me esters all bore a vicinal di-Me group toward the center of the chain. The assignment of the structure was confirmed by isolating one of the very long chain unusual fatty acids as the ester form after methanolysis and performing further analyses including 1H and 13C NMR spectroscopy and Fourier transform IR spectroscopy. Coupling this information with the data from gas chromatog./mass spectrometry anal., the exact

structure was confirmed as α,ω-15,16-dimethyltriacontanedioate di-Me ester. Addition of alcs., either metabolic (0.25M ethanol) or nonmetabolic (0.05M butanol) to cells grown at pH 7.0, or thermal stress (growth temperature at pH 7.0 was raised from 37 to 45 or 55°) also resulted in the synthesis of these very long chain fatty acids. Synthesis of these very long chain α,ω-dicarboxylic acids was reversed by reducing the temperature back to 37°. S. ventriculi is also unusual in that the membrane components are not the usual phospholipid components but appear to be predominantly glycolipids.
OS.CITING REF COUNT: 18 THERE ARE 18 CAPLUS RECORDS THAT CITE THIS RECORD (18 CITINGS)

L22 ANSWER 21 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1993:118712 HCAPLUS Full-text
DOCUMENT NUMBER: 118:118712
ORIGINAL REFERENCE NO.: 118:20489a,20492a
TITLE: Structure/activity investigations in eight arylalkyltriazenes comparison of chemical stability, mode of decomposition, and SCE induction in Chinese hamster V79-E cells
AUTHOR(S): Thust, Rudolf; Schneider, Martin; Wagner, Ursula; Schreiber, Dieter
CORPORATE SOURCE: Inst. Pathol. Anat., Med. Acad. Erfurt, Erfurt, O-5010, Germany
SOURCE: Cell Biology and Toxicology (1991), 7(2), 145-65
CODEN: CBTOE2; ISSN: 0742-2091
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A series of 7 1-aryl-3.3-dialkyltriazenes, including 1-phenyl-3.3-dimethyltriazene (DMPT), 1-phenyl-3.3-di(trideuteromethyl)triazene (DMPT-ds), 1-p-methylphenyl-3.3-dimethyltriazene (DMpMPT), 1-p-nitrophenyl-3.3-dimethyltriazene (DMpNPT), 1-phenyl-3.3-diethyltriazene (DEPT), 1-phenyl-3.3-di-n-propyltriazene (DnPrPT) and 1-phenyl-3.3-diisopropyltriazene (DiPrPT) and 1.3-diphenyl-3-methyltriazene (DPMT), was synthesized and characterized by UV/vis, IR and 1H NMR spectroscopy. Chemical half life was determined in phosphate buffer at 37° using UV/vis spectroscopy. With the exception of DMpNPT, which was stable, the triazenes underwent pH-dependent hydrolytic decomposition (acid catalysis). By means of UV/vis spectra, TLC and HPLC, phenol, aniline and secondary azocoupling products were identified after complete hydrolytic cleavage of the parent compds. Pathways of spontaneous hydrolysis are proposed and discussed. Genotoxic activity of the triazenes was assayed by measurement of sister chromatid exchanges (SCE) in V79-E cells without and with rat liver S9 mix as an exogenous metabolizing system. In the direct SCE assay (without S9 mix), all triazenes except DMpNPT exerted a toxic action (cell cycle delay) in a narrow concentration range between no effect and overt cytotoxicity. This non-specific toxicity depended on the pH of the incubation system and was inversely proportional to chemical half-life. The toxicity of these agents is most likely due to the arenediazonium cation which is a relatively stable intermediate. In a sublethal concentration range most triazenes induced significant increases of SCE rates. These are interpreted as an indirect consequence of cytotoxicity. Upon metabolic activation, the compds. were genotoxic in a dose-dependent fashion. Their SCE-inducing capacity depended on the nature of the alkylating species generated, i.e., the alkyldiazonium cation, and on chemical stability. Surprisingly, no deuterium isotope effect was observed in DMPT-d6. The order of genotoxic activity among the aryldialkyltriazenes was DMpNPT » DMPT = DMPT-ds > DMpMPT » DEPT > DnPrPT ≥ DiPrPT. DPMT was a marginal SCE inducer but very toxic upon metabolic activation. As monooxygenation of DPMT, like spontaneous hydrolysis, should generate a phenyldiazonium cation, the results suggest that arylation of DNA causes a very low SCE induction, if any.
OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD

L22 ANSWER 22 OF 66 HCAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1960:34267 HCAPLUS Full-text
DOCUMENT NUMBER: 54:34267
ORIGINAL REFERENCE NO.: 54:6716h-i,6717a-i,6718a-d
TITLE: Bisbenzylisoquinolines. III. Synthesis of isoquinoline intermediates
AUTHOR(S): Grundon, M. F.
CORPORATE SOURCE: Queen's Univ., Belfast, Ire.
SOURCE: Journal of the Chemical Society (1959) 3010-14
CODEN: JCSOA9; ISSN: 0368-1769
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB cf. C.A. 52, 17163a. Exploratory studies of an approach to the synthesis of unsym. cyclic bis(benzylisoquinoline) alkaloids, such as tubocurarine chloride, are described. 5'-Carboxymethyl-4-formyl-2,2'-dimethoxydiphenyl ether (9.65 g.), 5.0 g. rhodanine, and 10.0 g. anhydrous NaOAc is refluxed 30 min. in 50 ml. HOAc and the precipitated rhodanine derivative (I) faltered from the hot mixture; 1-hr. addnl. refluxing of the filtrate gives more I. Heating 13.1 g. I in 80 ml. 15% aqueous NaOH 40 min. on steam, cooling to 0°, and acidifying with dilute HCl gives the thiooxo acid, which is dried and refluxed 40 min. in 100 ml. EtOH containing NH2OH (from 8.6 g. NH2OH.HCl and 2.7 g. Na), the EtOH removed, 100 ml. H2O added to the residue, the resulting solution acidified with HCl, shaken with 40 ml. Et2O, and the hydroxyimino acid (II) (10.5 g.), m. 155-9°, filtered off. Concentration of the Et2O wash to 5 ml. gives 0.5 g. more II. Crude II (11 g.) in 50 ml. HOAc heated 20 min. on steam, the mixture diluted with 250 ml. H2O and let stand 12 hrs., and the precipitate triturated with EtOH gives 6.5 g. 5'-carboxymethyl-4-cyanomethyl-2,2'-dimethoxydiphenyl ether (III), m. 154-8°; treatment of mother liquors with CHCl3 yields 0.25 g. more III; recrystn. of III from EtOH gives rods, m. 158-60°. Shaking 2.2 g. III in 150 ml. HOAc containing 0.6 g. PtO2 with H 2.5 hrs. at room temperature and atmospheric pressure, removing the catalyst, evaporating the solution, and crystallizing the residue from EtOH gives 90% 4-(2-aminoethyl)-5'-carboxymethyl-2,2'-dimethoxydiphenyl ether (IV), m. 173-4°, strong absorption (in KBr) at 1570 cm.-1 (carboxy). Dissolved in 20 ml. MeOH containing 1 ml. concentrated H2SO4, kept 12 hrs., the solution evaporated, the residue taken up in H2O, and the solution treated with Na2CO3 and extracted with CHCl3, 50 mg. IV yields the corresponding Me ester (V) (oil), which is converted by heating 1 hr. at 140-50° with 30 mg. o-C6H4(CO)2O to 2,2'-dimethoxy-5'-methoxycarbonylmethyl-4-(2-phthalimidoethyl)diphenyl ether (VI), needles, m. 158-9° (EtOH), ν (KBr) 1764 and 1710 (N-phthaloyl) and 1736 (CO2Me) cm.-1 The corresponding free acid (VII), ν 1770 and 1716 cm.-1 (KBr) (Me2CO-MeOH), treated with CH2N2 in Et2O also gives VI. Refluxing 0.45 g. VII and 0.45 g. PCl5 10 min. in 10 ml. C6H6 and cooling gives 0.38 g. corresponding acid chloride (VIII), prisms, m. 142-4° (C6H6-petr. ether, b. 60-80°). Addition of 0.4 g. VIII in 10 ml. CHCl3 during 30 min. to 0.29 g. V and 0.25 g. Bu3N in 10 ml. CHCl3, after 12 hrs. washing the product with N HCl, N NaOH, and H2O, evaporating, triturating the residual gum with N HCl and H2O, and crystallizing from MeOH gives 0.6 g. N-{4-methoxy-3-[2-methoxy-4-(2- phthalimidoethyl)phenoxy]phenyl}acetyl-2-[3-methoxy-4-(2-methoxy-5- methoxycarbonylmethyl)phenoxy]ethylamine (IX), fine needles, m. 80-1°, ν (KBr) 1768 and 1705 (N-phthaloyl), 1743 (CO2Me), and 1673 (amide) cm.-1 Addition of 0.35 g. VIII in aqueous dioxane during 45 min. to 0.17 g. IV and 0.03 g. Na2CO3 in the same solvent, maintaining alkalinity by periodic addition of aqueous Na2CO3, and after an addnl. hr. acidifying with HCl and extracting with CHCl3 gives a gum (0.35 g.), ν 1775, 1710, and 720 cm.-1 (KBr), which on esterification with CH2N2 also gives IX, in poor overall

yield. Refluxing 0.37 g. IX 30 min. with 2 ml. POCl3 in 20 ml. PhMe, evaporating, adding H2O to the residue, extracting with CHCl3, and triturating the residue from the extract with PhMe yields the crude hydrochloride of 3,4-dihydro-6-methoxy-7-(2-methoxy-5- methoxycarbonylmethylphenoxy)-1-{4-methoxy-3-[2-methoxy-4-(2- phthalimidoethyl)phenoxy]benzyl}isoquinoline (X), ν (of X.HCl) 1770 and 1710 (N-phthaloyl) and 1650 (C:NH+) cm.-1; dissolving the crude salt in 30 ml. warm EtOH, cooling, filtering off the insol. material, and adding excess picric acid in EtOH gives 0.34 g. X picrate, yellow prisms, m. 178-9° (MeOH). In the process of shaking 90 mg. X picrate in CHCl3 with several portions aqueous Na2CO3, the liberated base (X) is autoxidized to the corresponding benzoyl compound (XI), needles, m. 101-3° (EtOH), ν 1765 and 1708 (N-phthaloyl), 1730 (CO2Me), and 1667 (COC:N) cm.-1; picrate m. 110-14° (EtOH). Heating XI in Ac2O gives a blue-green color. Refluxing X (from 270 mg. picrate) with MeI in MeOH 1.5 hrs. and evaporating leaves crude yellow X methiodide (XIII), m. 110-14°, ν 1770 and 1715 cm.-1 (assigned to N-phthaloyl group). Hydrogenation of crude XII over 0.5 PtO2 in 100 ml. EtOH containing 0.5 ml. Et2NH at room temperature and atmospheric pressure, removal of catalyst and solvent, shaking the residue (in CHCl3) with 2N NaOH and H2O, evaporating the CHCl3 solution, and triturating the residue with 40-60° petr. ether leaves 160 mg. solid, converted (in EtOH) to 1,2,3,4-tetrahydro-6-methoxy-7-(2-methoxy-5-methoxycarbonylmethylphenoxy)- 1-{4-methoxy-3-[2-methoxy-4-(2-phthalimidoethyl)phenoxy]benzyl}2- methylisoquinoline picrate (180 mg.), m. 103-5° (hydrate) (EtOH), ν (of free base) 1757 and 1700 (N-phthaloyl) and 1725 (CO2Me) cm.-1 Action of NaBH4 on XII gives a product which, on the basis of infrared examination, ν 1735 (CO2Me), 1700 (CO2H), and 1685 (amide) cm.-1, has apparently suffered partial hydrolysis of the phthaloyl group. Similarly, addition of 50 mg. NaBH4 in portions to 50 mg. VI in 30 ml. MeOH and, after 10 min., removal of MeOH, addition of H2O, acidification with HCl, and extraction with CHCl3 gives 25 mg. product, prisms, m. 179-80° (EtOH), undepressed by mixture with VII, but having different infrared absorption, ν 1720 and 1700 (CO2H), and 1667 (amide) cm.-1 Heating 1 g. 2-(3,4-dimethoxyphenyl)ethylamine with 0.9 g. o-C6H4(CO)2O in 10 ml. HOAc 2 hrs. on a steam bath, diluting the mixture with H2O, and recrystg. the precipitate from MeOH gives the corresponding phthalimide (XIII), plates, m. 173-4°, ν 1755 and 1700 (N-phthaloyl) cm.-1 XIII is unchanged by action of NaBH4, but hot aqueous ethanolic N NaOH converts it to the corresponding phthalamic acid, rectangular plates, m. 171-2° (EtOH) (m.p. with XIII undepressed), ν 1718 (CO2H) and 1625 (amide) cm.-1 PCl5 (300 mg.) is added to 280 mg. IX in 10 ml. CHCl3, the mixture kept 3 days at room temperature, then shaken with H2O, the CHCl3 phase evaporated, and the residue crystallized from MeOH to give 180 mg. C45H43O10N2Cl.H2O, m. 100-2°, the Cl in which is inert to cold NaOH. Refluxing 0.75 g. III in 7 ml. SOCl2 2 hrs., evaporating, adding the residue (in 20 ml. CHCl3) during 30 min. to 0.75 g. V and 0.45 g. Bu3N in 20 ml. CHCl3, after letting the mixture stand overnight evaporating, triturating the residue with N HCl, dissolving in CHCl3, shaking with aqueous Na2CO3, and removing solvent gives a crude amide (gum, 1.15 g.), which is refluxed 45 min. with 3 ml. POCl3 in 20 ml. PhMe, the solution evaporated, the residue boiled with 60 ml. EtOH, and the clear supernatant from the cooled mixture treated with excess picric acid in EtOH to give 0.91 g. crude 3,4-dihydro-6-methoxy-1-{4-methoxy-3-[4-(2-cyanoethyl)- 2-methoxyphenoxy]benzyl}-7-(2-methoxy-5- methoxycarbonylmethylphenoxy)isoquinoline (XIV) picrate, m. 89-92°, from which the pure substance is extracted with 60 ml. boiling MeOH and recrystd. from Me2CO-MeOH, yellow needles, m. 119-20°. Nothing characterizable is obtained by reduction of XIV.

L22 ANSWER 23 OF 66 MEDLINE on STN
ACCESSION NUMBER: 2009593414 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 19720028

TITLE: Sphingosine-1-phosphate as an amphipathic metabolite: its properties in aqueous and membrane environments.
AUTHOR: Garcia-Pacios Marcos; Collado M Isabel; Busto Jon V; Sot Jesus; Alonso Alicia; Arrondo Jose-Luis R; Goni Felix M
CORPORATE SOURCE: Unidad de Biofisica (CSIC-UPV/EHU), Universidad del Pais Vasco, Bilbao, Spain.
SOURCE: Biophysical journal, (2009 Sep 2) Vol. 97, No. 5, pp. 1398-407.
Journal code: 0370626. E-ISSN: 1542-0086. L-ISSN: 0006-3495.
Report No.: NLM-PMC2749770 [Available on 09/02/10].
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200912
ENTRY DATE: Entered STN: 2 Sep 2009
Last Updated on STN: 16 Dec 2009
Entered Medline: 7 Dec 2009

AB Sphingosine-1-phosphate (S1P) is currently considered to be an important signaling molecule in cell metabolism. We studied a number of relevant biophysical properties of S1P, using mainly Langmuir balance, differential scanning calorimetry, (31)P-NMR, and infrared (IR) spectroscopy. We found that, at variance with other, structurally related sphingolipids that are very hydrophobic, S1P may occur in either an aqueous dispersion or a bilayer environment. S1P behaves in aqueous media as a soluble amphiphile, with a critical micelle concentration of approximately 12 muM. Micelles give rise to larger aggregates (in the micrometer size range) at and above a 1 mM concentration. The aggregates display a thermotropic transition at approximately 60 degrees C, presumably due to the formation of smaller structures at the higher temperatures. S1P can also be studied in mixtures with phospholipids. Studies with dielaidoylphosphatidylethanolamine (DEPE) or deuterated dipalmitoylphosphatidylcholine (DPPC) show that S1P modifies the gel-fluid transition of the glycerophospholipids, shifting it to lower temperatures and decreasing the transition enthalpy. Low (<10 mol %) concentrations of S1P also have a clear effect on the lamellar-to-inverted hexagonal transition of DEPE, i.e., they increase the transition temperature and stabilize the lamellar versus the inverted hexagonal phase. IR spectroscopy of natural S1P mixed with deuterated DPPC allows the independent observation of transitions in each molecule, and demonstrates the existence of molecular interactions between S1P and the phospholipid at the polar headgroup level that lead to increased hydration of the carbonyl group. The combination of calorimetric, IR, and NMR data allowed the construction of a temperature-composition diagram ("partial phase diagram") to facilitate a comparative study of the properties of S1P and other related lipids (ceramide and sphingosine) in membranes. In conclusion, two important differences between S1P and ceramide are that S1P stabilizes the lipid bilayer structure, and physiologically relevant concentrations of S1P can exist dispersed in the cytosol.

L22 ANSWER 24 OF 66 MEDLINE on STN
ACCESSION NUMBER: 2009108119 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 19066942
TITLE: Remodelling of arabinoxylan in wheat (Triticum aestivum) endosperm cell walls during grain filling.
AUTHOR: Toole G A; Barron C; Le Gall G; Colquhoun I J; Shewry P R; Mills E N C

CORPORATE SOURCE: Institute of Food Research, Colney, Norwich, NR4 7UA, UK.. geraldine.toole@bbsrc.ac.uk
CONTRACT NUMBER: (United Kingdom Biotechnology and Biological Sciences Research Council)
SOURCE: Planta, (2009 Feb) Vol. 229, No. 3, pp. 667-80. Electronic Publication: 2008-12-09.
Journal code: 1250576. ISSN: 0032-0935.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200907
ENTRY DATE: Entered STN: 3 Feb 2009
Last Updated on STN: 15 Jul 2009
Entered Medline: 14 Jul 2009

AB Previous studies using spectroscopic imaging have allowed the spatial distribution of structural components in wheat endosperm cell walls to be determined. FT-IR microspectroscopy showed differing changes in arabinoxylan (AX) structure, during grain development under cool/wet and hot/dry growing conditions, for differing cultivars (Toole et al. in Planta 225:1393-1403, 2007). These studies have been extended using Raman microspectroscopy, providing more details of the impact of environment on the polysaccharide and phenolic components of the cell walls. NMR studies provide complementary information on the types and levels of AX branching both early in development and at maturity. Raman microspectroscopy has allowed the arabinose:xylose (A/X) ratio in the cell wall AX to be determined, and the addition of ferulic acid and related phenolic acids to be followed. The changes in the A/X ratio during grain development were affected by the environmental conditions, with the A/X ratio generally being slightly lower for samples grown under cool/wet conditions than for those from hot/dry conditions. The degree of esterification of the endosperm cell walls with ferulic acid was also affected by the environment, being lower under hot/dry conditions. The results support earlier suggestions that AX is either delivered to the cell wall in a highly substituted form and is remodelled through the action of arabinoxylan arabinofuranohydrolases or arabinofuranosidases, or that low level substituted AX are incorporated into the wall late in cell wall development, reducing the average degree of substitution, and that the rate of this remodelling is influenced by the environment. (1)H NMR provided a unique insight into the chemical structure of intact wheat endosperm cell walls, providing qualitative information on the proportions of mono- and disubstituted AX and the levels of branching of adjacent units. The A/X ratio did not change greatly with either the development stage or the growth conditions, but the ratio of mono- to disubstituted Xylp residues increased markedly (by about fourfold) in the more mature samples, confirming the changes in branching levels determined using FT-IR. To the best of our knowledge, this is the first time that intact endosperm cell walls have been studied by (1)H NMR.

L22 ANSWER 25 OF 66 MEDLINE on STN
ACCESSION NUMBER: 2005305704 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 15952788
TITLE: Influence of the 33 kDa manganese-stabilizing protein on the structure and substrate accessibility of the oxygen-evolving complex of photosystem II.
AUTHOR: Gregor Wolfgang; Cinco Roehl M; Yu Hui; Yachandra Vittal K; Britt R David
CORPORATE SOURCE: Department of Chemistry, University of California, Davis, California 95616, USA. Wolfgang.Gregor@ vu-wien.ac.at.
CONTRACT NUMBER: GM48242 (United States NIGMS NIH HHS)

GM55302 (United States NIGMS NIH HHS)
SOURCE: Biochemistry, (2005 Jun 21) Vol. 44, No. 24, pp. 8817-25.
Journal code: 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200509
ENTRY DATE: Entered STN: 15 Jun 2005
Last Updated on STN: 27 Sep 2005
Entered Medline: 26 Sep 2005

AB The 33 kDa manganese-stabilizing extrinsic protein binds to the lumenal side of photosystem II (PS II) close to the Mn(4)Ca cluster of the oxygen-evolving complex, where it limits access of small molecules to the metal site. Our previous finding that the removal of this protein did not alter the magnetic coupling regime within the manganese cluster, measured by electron spin-echo envelope modulation [Gregor, W., and Britt, R. D. (2000) Photosynth. Res. 65, 175-185], prompted us to examine whether this accessibility control is also true for substrate water, using the same pulsed EPR technique. Comparing the deuteron modulation of the S(2)-state multiline signal of PS II membranes, equilibrated with deuterated water (D(2)O) after removal or retention of the 33 kDa protein, we observed no change in the number and the distance of deuterons magnetically coupled to manganese, indicating that the number and distance of water molecules bound to the manganese cluster are independent of bound 33 kDa protein in the S(1) state, in which the sample was poised prior to cryogenic illumination. A simple modulation depth analysis revealed a distance of 2.5-2.6 A between the closest deuteron and manganese. These results are in agreement with our refined X-ray absorption analysis. The manganese K-edge positions, reflecting their oxidation states, and the extended X-ray absorption fine structure amplitudes and distances between the manganese ions and their oxygen and nitrogen ligands (1.8, 2.7, and 3.3-3.4 A) were independent of bound 33 kDa protein.

L22 ANSWER 26 OF 66 MEDLINE on STN
ACCESSION NUMBER: 2002422217 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 12177179
TITLE: Synthesis and biochemical properties of a new photoactivatable cholesterol analog 7,7-azocholestanol and its linoleate ester in Chinese hamster ovary cell lines.
AUTHOR: Cruz Jonathan C; Thomas Matthew; Wong Edmund; Ohgami Nobutaka; Sugii Shigeki; Curphey Thomas; Chang Catherine C Y; Chang Ta-Yuan
CORPORATE SOURCE: Department of Biochemistry, Medical School, Dartmouth College, Hanover, NH, USA.
CONTRACT NUMBER: HL 36709 (United States NHLBI NIH HHS)
HL 60306 (United States NHLBI NIH HHS)
SOURCE: Journal of lipid research, (2002 Aug) Vol. 43, No. 8, pp. 1341-7.
Journal code: 0376606. ISSN: 0022-2275.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304

ENTRY DATE: Entered STN: 15 Aug 2002
Last Updated on STN: 6 Apr 2003
Entered Medline: 4 Apr 2003

AB We report the chemical synthesis of a new photoactivatable cholesterol analog 7,7-azocholestanol (AC) and its linoleate ester (ACL). We also examined the biochemical properties of the sterol and its ester by employing several different mutant Chinese hamster ovary (CHO) cell lines with defined abnormalities in cholesterol metabolism as tools. AC mimics cholesterol in supporting the growth of a mutant cell line (M19) that requires cholesterol for growth. In normal cells, tritiated ACL present in low-density lipoprotein (LDL) was hydrolyzed and reesterified in a manner similar to tritiated cholesteryl linoleate (CL) in LDL. Also, in the mutant cell line (AC29) lacking the enzyme acyl-coenzyme A:cholesterol acyltransferase or in the mutant cell line (CT60) defective in the Niemann-Pick type C1 protein, the hydrolysis of ACL in LDL was normal, but the reesterification of the liberated AC was defective. Therefore, the metabolism of ACL in LDL is very similar to that of CL in LDL. Tritium-labeled AC delivered to intact CHO cells as a cyclodextrin complex was shown to photoaffinity label several discrete polypeptides, including caveolin-1. These results demonstrate AC as an effective reagent for studying cholesterol-protein interactions involved in intracellular cholesterol trafficking.

L22 ANSWER 27 OF 66 MEDLINE on STN
ACCESSION NUMBER: 1999017882 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 9799489
TITLE: Conformational and dynamic changes of Yersinia protein tyrosine phosphatase induced by ligand binding and active site mutation and revealed by H/D exchange and electrospray ionization Fourier transform ion cyclotron resonance mass spectrometry.
AUTHOR: Wang F; Li W; Emmett M R; Hendrickson C L; Marshall A G; Zhang Y L; Wu L; Zhang Z Y
CORPORATE SOURCE: Department of Molecular Pharmacology, Albert Einstein College of Medicine, Bronx, New York 10461, USA.
CONTRACT NUMBER: CA 69202 (United States NCI NIH HHS)
GM-31683 (United States NIGMS NIH HHS)
SOURCE: Biochemistry, (1998 Nov 3) Vol. 37, No. 44, pp. 15289-99.
Journal code: 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 15 Jan 1999
Last Updated on STN: 3 Mar 2000
Entered Medline: 30 Nov 1998

AB Protein tyrosine phosphatases (PTPase) play important roles in the intracellular signal transduction pathways that regulate cell transformation, growth, and proliferation. Here, solvent accessibility is determined for backbone amide protons from various segments of wild-type Yersinia PTPase in the presence or absence of 220 microM vanadate, a competitive inhibitor, as well as an active site mutant in which the essential cysteine 403 has been replaced by serine (C403S). The method consists of solution-phase H/D exchange, followed by pepsin digestion, high-performance liquid chromatography, and electrospray ionization high-field (9.4 T) Fourier

transform ion cyclotron resonance mass spectrometry. Proteolytic segments spanning approximately 93.5% of the primary sequence are analyzed. Binding of vanadate reduces the H/D exchange rate throughout the protein, both for the WpD loop and for numerous other residues that are shielded when that loop is pulled down over the active site on binding of the inhibitor. The single active site C403S mutation reduces solvent access to the WpD loop itself, but opens up the structure in several other segments. Although the 3D structure of the ligand-bound C403S mutant is similar to that of the wild-type PTPase, and the C403S mutant and the wild-type enzyme display similar affinities for vanadate, the thermodynamics for binding of vanadate is different for the two proteins. Collectively, these results establish the flexibility of the WpD loop (previously inferred by comparing PTPase X-ray single-cyrstal diffraction structures in the presence and absence of a tungstate inhibitor), as well as several other signficant changes in segment exposure and/or flexibility that are not evident from X-ray structures.

L22 ANSWER 28 OF 66 MEDLINE on STN
ACCESSION NUMBER: 1992117162 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 1662914
TITLE: D2O as a substitute for 3H2O, as a reference indicator in liver multiple-indicator dilution studies.
AUTHOR: Pang K S; Xu N; Goresky C A
CORPORATE SOURCE: Faculty of Pharmacy, University of Toronto, Ontario, Canada.
CONTRACT NUMBER: GM-38250 (United States NIGMS NIH HHS)
SOURCE: The American journal of physiology, (1991 Dec) Vol. 261, No. 6 Pt 1, pp. G929-36. Journal code: 0370511. ISSN: 0002-9513.
PUB. COUNTRY: United States
DOCUMENT TYPE: (COMPARATIVE STUDY) Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T) (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199202
ENTRY DATE: Entered STN: 8 Mar 1992 Last Updated on STN: 3 Feb 1997 Entered Medline: 14 Feb 1992

AB The hepatic space of distribution and the processes underlying uptake of tracer substrate may be appraised by the multiple-indicator dilution technique after simultaneous injection of noneliminated vascular (51Cr-labeled red blood cells), extracellular 125I-labeled albumin and [14C]sucrose (or [58Co]EDTA) as high- and low-molecular-weight interstitial references, respectively], and cellular (3H2O or [14C]urea) indicators, together with the tracer-labeled substrate. The use of 3H2O or [14C]urea, with [14C]- or [3H]sucrose, however, precludes the simultaneous introduction and analysis of the behavior of 3H- and 14C-labeled substrate and metabolite. An assay for the quantitation of D2O in plasma by Fourier transform infrared spectrometry was therefore developed such that D2O could be used in lieu of 3H2O in multiple-indicator dilution studies in the blood-perfused rat liver. In experiments performed with an injection dose containing 51Cr-labeled red blood cells, 125I-labeled albumin, [14C]sucrose, 3H2O, and D2O, D2O was found to behave virtually identical to 3H2O in blood and liver; the accessible cellular water spaces were 0.625 and 0.621 ml/g liver for 3H2O and D2O, respectively, and the corresponding ratios of the sum of the cellular water plus the interstitial water space to the sinusoidal water space were 3.87 and 3.89. D2O was found to be an ideal substitute and is much superior to [14C]urea, which exhibits a

small red blood cell carriage effect and which is slightly less dispersed than 3H2O.

L22 ANSWER 29 OF 66 MEDLINE on STN
ACCESSION NUMBER: 1991268103 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 1828807
TITLE: Cytotoxicity of a BIS-GMA dental composite before and after leaching in organic solvents.
AUTHOR: Rathbun M A; Craig R G; Hanks C T; Filisko F E
CORPORATE SOURCE: W.R. Grace and Company, Washington Research Center, Columbia, Maryland 21044.
CONTRACT NUMBER: 5-T32-DE07057 (United States NIDCR NIH HHS)
SOURCE: Journal of biomedical materials research, (1991 Apr) Vol. 25, No. 4, pp. 443-57.
Journal code: 0112726. ISSN: 0021-9304.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199107
ENTRY DATE: Entered STN: 11 Aug 1991
Last Updated on STN: 3 Mar 2000
Entered Medline: 19 Jul 1991
AB Cell culture techniques were used to determine the source of cytotoxic agents in a commercial BIS-GMA composite. The material was polymerized according to the manufacturer's directions and leachable components were removed by room temperature extraction in ethanol, chloroform, or toluene. The leachable components in the extracts were identified using infrared spectrographic analysis. Thin layer chromatographic analysis was used to determine the number of constituents. These constituents were separated by gas chromatography and then identified by mass spectrographic analysis. Succinic dehydrogenase activity and radioactive labeling with tritiated leucine were used to evaluate cell metabolism and protein synthesis, respectively. The infrared analysis of the extracts showed that the primary component was unreacted BIS-GMA. Trace amounts of 2-hydroxy-4-methoxy-benzophenone, a light stabilizer, as well as a phenyl ester of benzoic acid which was probably degraded from BIS-GMA, were detected by the mass spectrographic method. The removal of leachable components caused a 90% decrease in toxicity compared to the nonextracted BIS-GMA samples. The extracted BIS-GMA samples showed no cellular response compared to the Teflon negative control.

L22 ANSWER 30 OF 66 MEDLINE on STN
ACCESSION NUMBER: 1990336912 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 1696215
TITLE: Mutagenicity and cytotoxicity of N-nitrosothiazolidine-4-carboxylic acid.
AUTHOR: Lin I N; Gruenwedel D W
CORPORATE SOURCE: Department of Food Science and Technology, University of California, Davis 95616.
SOURCE: Food additives and contaminants, (1990 May-Jun) Vol. 7, No. 3, pp. 357-68.
Journal code: 8500474. ISSN: 0265-203X.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 199009
ENTRY DATE: Entered STN: 12 Oct 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 11 Sep 1990

AB N-Nitrosothiazolidine-4-carboxylic acid (NTCA) was prepared by treating L-thioproline with sodium nitrite at pH 2, 37 degrees C. The compound was characterized by mass spectrometry, infrared spectroscopy and 1H-nuclear magnetic resonance spectroscopy. The cytotoxic and mutagenic properties of NTCA were explored by exposing the human cell line HeLa S3 at 37 degrees C to various concentrations (10 microM-10 mM) of the compound for various periods of time (1-36 h) and by monitoring its effects on cell viability, cell growth, intracellular metabolic activities such as DNA, RNA, and protein synthesis and on DNA repair synthesis ('unscheduled' DNA synthesis). NTCA did not affect the cells' viability at any concentration or incubation period but decreased cell growth at the limiting concentration of 10 mM in the growth medium. NTCA had no effect on RNA and protein synthesis, and, similarly, it had no effect on DNA synthesis at concentrations up to 3 mM. Curiously, the stimulation of DNA synthesis by NTCA was seen at 10 mM after 24 h of incubation. NTCA did not initiate 'unscheduled' DNA synthesis (DNA repair). It is concluded that the compound displays very little cytotoxicity and no mutagenicity in the HeLa S3 test system; hence, its presence in humans and in the human food supply is likely to be of little importance as far as its oncogenic properties are concerned.

L22 ANSWER 31 OF 66 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2007:492987 BIOSIS Full-text
DOCUMENT NUMBER: PREV200700498990
TITLE: Biocatalysis of the anticancer sipholane triterpenoids.
AUTHOR(S): Jain, Sandeep; Shirode, Arnit; Yacoub, Shenouda; Barbo, Ashley; Sylvester, Paul W.; Huntimer, Eric; Halaweish, Fathi; El Sayed, Khalid A. [Reprint Author]
CORPORATE SOURCE: NE Louisiana Univ, Coll Pharm, Dept Basic Pharmaceut Sci, 700 Univ Ave, Monroe, LA 71209 USA
elsayed@ulm.edu
SOURCE: Planta Medica, (JUN 2007) Vol. 73, No. 6, pp. 591-596.
CODEN: PLMEAA. ISSN: 0032-0943.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Sep 2007
Last Updated on STN: 20 Sep 2007

AB The Red Sea sponge Callyspongia (= Siphonochalina) siphonella is a rich source of sipholane triterpenoids. Biocatalysis of the major sipholanes, sipholenol A (1) and sipholenone A (2), respectively, by Mucor ramannianus ATCC 9628 and Cunninghamella elegans ATCC 7929 afforded four new metabolites 3 - 6 along with sipholenol G and 28-hydroxysipholenol A. Major sipholanes along with their biocatalytic products were investigated for their antiproliferative activity against the highly malignant +SA mouse mammary epithelial cell line. Sipholenone A (2) was the most active sipholane inhibiting +SA cell proliferation with an IC50 value of 20-30 mu M. Sipholenone A, also, showed cytotoxicity against MCF-7 at a dose of 0.9 mu M and antiangiogenic activity in the CAM (chorio-allantoic membrane) assay. This is the first report on anticancer activity of these triterpenoids.

L22 ANSWER 32 OF 66 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:441154 BIOSIS Full-text
DOCUMENT NUMBER: PREV200400441527

TITLE: 10th International Symposium on Urolithiasis, Hong Kong, China, May 25-28, 2004.
AUTHOR(S): Anonymous
SOURCE: Urological Research, (May 2004) Vol. 32, No. 2, pp. 135-173. print.
Meeting Info.: 10th International Symposium on Urolithiasis. Hong Kong, Hong Kong, China. May 25-28, 2004.
CODEN: URLRA5. ISSN: 0300-5623.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Summary)
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Nov 2004
Last Updated on STN: 17 Nov 2004

AB This meeting on the clinical and non-clinical studies of urolithiasis in humans and non-humans contains 159 abstracts written in English. The abstracts are divided into 10 specific categories, including: Physical Chemistry: Supersaturation and Crystallization; Crystallization Modulators and Macromolecules; Cell/Tissue - Crystal Interaction; Pathophysiology and Metabolic Disorders; Genetic Disorders and Pediatric Stone Disease; Uric Acid Stones; Medical and Surgical Management of Renal Stones; Nutrition, Dietary Risk Factors and Water Intake; Epidemiology and Economics; and Animal Urolithiasis. Topics include the use of the Bonn-Risk-Index in urine samples, a new method using a chemiluminescence analysis system to measure urinary oxalate and citrate, a comparison of the Fourier transmission infra red analysis (FTIR) against optical microscopy in urinary stone analysis, a study of protein modulators of urolithiasis in Pakistani pediatric stone-formers, the potential role of fibronectin in stone formation, the association of nephrolithiasis and metabolic syndrome, and the usefulness of extracorporeal shock wave lithotripsy (ESWL) for treating canine and feline ureteroliths.

L22 ANSWER 33 OF 66 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:59765 BIOSIS Full-text
DOCUMENT NUMBER: PREV200300059765
TITLE: Carbon mass balance evaluation of cellulase production on soluble and insoluble substrates.
AUTHOR(S): Saez, Juan Carlos [Reprint Author]; Schell, Daniel J.; Tholudur, Arun; Farmer, Jody; Hamilton, Jenny; Colucci, Jose A.; McMillan, James D.
CORPORATE SOURCE: Department of Chemical Engineering, University of Puerto Rico, Mayaguez Campus, P.O. Box 9046, Mayaguez, 00681-9046, Puerto Rico
juan_carlos_saez@nrel.gov
SOURCE: Biotechnology Progress, (November-December 2002) Vol. 18, No. 6, pp. 1400-1407. print.
CODEN: BIPRET. ISSN: 8756-7938.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Jan 2003
Last Updated on STN: 22 Jan 2003

AB A methodology is described and applied for performing carbon mass balances across cellulase enzyme production processes using both soluble sugar and insoluble cellulose substrates. The fungus Trichoderma reesei was grown on either glucose, lactose, or cellulose in aerobic batch mode, and the evolution of the main carbonaceous components (cell mass, cellulose, soluble protein, adsorbed protein, sugars, and carbon dioxide) was followed. A variety of analytical techniques were utilized to measure these components, including (i) gravimetric analysis, (ii) near-infrared spectroscopy, (iii) bicinchoninic acid based soluble protein measurement, (iv) gas mass spectrometry and flow

rate, (v) CHNS/O elemental analyses, and (vi) high-performance liquid chromatography. The combined set of measurements allowed carbon mass balances across the cellulase production process to be assessed to determine the consistency of the underlying kinetic data. Results demonstrate the capability to determine the levels and distribution of all major carbonaceous components during the cellulase production process on both soluble and insoluble substrates. Average carbon mass balance closures were near 100% during early stages (<72 h) of the cultivations using glucose, lactose, or cellulose as the substrates, but carbon mass closures trended high later in the cultivation. Analysis of carbon allocation results suggests that an error in the gas mass flow rate measurement was the primary cause for carbon mass balance closures to exceed 110% late in the process.

L22 ANSWER 34 OF 66 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1996:574671 BIOSIS Full-text
DOCUMENT NUMBER: PREV199799289352
TITLE: Investigation of poly(beta-L-malic acid) production by strains of Aureobasidium pullulans.
AUTHOR(S): Liu, S.; Steinbuechel, A. [Reprint author]
CORPORATE SOURCE: Inst. Mikrobiol., Westfaelische Wilhelms-Univ. Muenster, Corrensstrasse 3, D-48149 Muenster, Germany
SOURCE: Applied Microbiology and Biotechnology, (1996) Vol. 46, No. 3, pp. 273-278. CODEN: AMBIDG. ISSN: 0175-7598.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Dec 1996
Last Updated on STN: 23 Dec 1996

AB Eight strains of the genus Aureobasidium obtained from culture collections were tested for their capability to produce poly(beta-L-malic acid) (PMA). Four of the tested strains showed positive results. The most productive strain, A. pullulans CBS 591.75, was used to study the production of PMA in stirred-tank reactors. It was found that PMA was mainly produced in the late exponential phase, and the production related positively to glucose consumption. At the beginning of the fermentation the pH increased from 4.0 to about 7.0; subsequently the pH decreased and remained stable at around 3.0-3.5 for several days. Temperatures higher than 25 degree C were detrimental to PMA production and cell growth. PMA production and cell growth at 20 degree C and 25 degree C exhibited no significant differences. PMA production and cell growth were studied under pH-controlled fermentation (at pH 2.0,4.0,5.5). The highest PMA production occurred at pH 4.0. PMA production was reduced at pH 2.0 although quite reasonable cell growth occurred at this pH value. Under optimized conditions 9.8 g PMA/l was produced during 9 days of fermentation in the stirred-tank reactors with an overall yield of 0.11 g PMA/g glucose. A procedure for the isolation of PMA and its separation from the other components of the fermentation broth was developed. The isolated PMA was characterized by 1H and 13C-NMR spectroscopy as well as by infrared absorption spectroscopy. Gel-permeation chromatography revealed a relative molecular mass of approximately 3000-5000 by comparison with polyethylene glycol standards.

L22 ANSWER 35 OF 66 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1994:313998 BIOSIS Full-text
DOCUMENT NUMBER: PREV199497326998
TITLE: Microbial oxidation of cumene by octane-grown cells.

AUTHOR(S): Hou, C. T. [Reprint author]; Jackson, M. A.; Bagby, M. O.; Becker, L. A.
CORPORATE SOURCE: Oil Chem. Res., Natl. Cent. Agric. Utilization Res., Agric. Res. Serv., U.S. Dep. Agric., 1815 North University St., Peoria, IL 61604, USA
SOURCE: Applied Microbiology and Biotechnology, (1994) Vol. 41, No. 2, pp. 178-182.
CODEN: AMBIDG. ISSN: 0175-7598.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 26 Jul 1994
Last Updated on STN: 27 Jul 1994

AB Previously, we reported that eight glucose-grown microbial cultures out of 1229 screened oxidize the alkyl side-chain of 2-phenylpropane (cumene) stereospifically. Now, we have adapted these cultures to grow on n-octane and found that their cumene oxidation activities increased more than 30 times. We also found an additional 11 cultures (ten bacteria, one actinomycete) that oxidized cumene when grown on octane but not on glucose. In general, octane-grown cells were more active in cumene oxidation than glucose- grown cells. Rhodococcus rhodochrous NRRL B-2153 showed the best conversion yield (2-phenyl-1-propanol plus 2-phenyl-1-propionic acid was 5.5%) at 25 degree C, pH 8.0, 250 rpm, and 12 h of reaction. Structures of the reaction products were confirmed by gas chromatography (GC)/mass spectrometry and GC/infrared analyses. Products contained 84% ee (enantiomeric excess) of the R(-) isomer, as analyzed with a GC cyclodextrin chiral column. Strain B-2153 oxidized alkylbenzenes in the following order of reaction rate: ethylbenzene gt amylbenzene gt butylbenzene gt cumene gt propyl-benzene gt sec-butylbenzene. tert-Butylbenzene was not oxidized.

L22 ANSWER 36 OF 66 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1991:47986 BIOSIS Full-text
DOCUMENT NUMBER: PREV199191026267; BA91:26267
TITLE: PORPHYRA CELL CULTURES ISOLATION GROWTH AND POLYSACCHARIDE PRODUCTION.
AUTHOR(S): TAIT M I [Reprint author]; MILNE A M; GRANT D; SOMERS J A; STAPLES J; LONG W F; WILLIAMSON F B; WILSON S B
CORPORATE SOURCE: DEP MOLECULAR CELL BIOL, UNIV ABERDEEN, MARISCHAL COLL, ABERDEEN AB9 1AS, SCOTLAND, UK
SOURCE: Journal of Applied Phycology, (1990) Vol. 2, No. 1, pp. 63-70.
CODEN: JAPPEL. ISSN: 0921-8971.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 10 Jan 1991
Last Updated on STN: 10 Jan 1991

AB A range of cell lines was isolated from Porphyra umbilicalis L. (Rhodophyta) tissue using a variety of methods, the most successful involving exposure to a limpet acetone powder enzyme extract for 24 h, homogenisation and filtration through a series of polyester meshes. All established lines grew as 0.1-5 mm diameter aggregates in liquid culture; most were stable and have been grown in shake-flask or air-lift culture for periods in excess of 1 yr without reverting to the foliose growth form. An investigation of the medium used to grow these lines indicated that it was not nitrogen-deficient and that the sodium chloride concentration was optimal. The addition of an organic buffer increased the final cell yield. None of these cell lines grew heterotrophically in medium supplemented with a range of fixed carbon sources. The infrared spectra of polysaccharides isolated from Porphyra aggregates and

from tissue grown under identical conditions indicated that the structures of the two isolates were analogous.

L22 ANSWER 37 OF 66 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1978:149049 BIOSIS Full-text
DOCUMENT NUMBER: PREV197865036049; BA65:36049
TITLE: THE INFLUENCE OF PRE TREATMENT WITH DIFFERENT CATIONS ON ANAEROBIC NITRITE PRODUCTION BY EXCISED PISUM-SATIVUM ROOTS.
AUTHOR(S): SAHULKA J [Reprint author]
CORPORATE SOURCE: INST EXP BOT, CZECH ACAD SCI, KE DVORU 16/15, 166 30 PRAHA 6, CZECH
SOURCE: Biologia Plantarum (Prague), (1977) Vol. 19, No. 5, pp. 338-345.
CODEN: BPABAJ. ISSN: 0006-3134.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB The influence of pretreatment with some cations on anaerobic nitrite production (in an assay medium lacking nitrate) by excised primary roots of pea (P. sativum L. cv. Raman), detached from 6 day old seedlings germinated in distilled water, was investigated. When the excised roots were precultivated in one-salt-solutions of KNO3, they produced, at 9 mM and 15 mM NO3- concentrations under anaerobic conditions, significantly more NO2- than those precultivated in a nutrient solution containing K+, Ca2+ and Mg2+, and they produced nitrite for a longer time. The KNO3 dependent increase in anaerobic NO2- production was counteracted most by Ca2+ and to a lesser extent by Mg2+; Na+ was without effect. NH4+ at higher concentrations (12 and 15 mM) significantly depressed nitrite production both by roots precultivated in a solution containing NH4+ and K+, and by roots precultivated in a full nutrient solution containing K+, Ca2+ and Mg2+. At lower NH4+ concentrations (0.6 and 2 mM NH4+; 15 mM NO3-) the decrease was more conspicuous in the KNO3 solution than in the full nutrient solution. Nitrate reductase level was not influenced by this pretreatment. When 5% and 7.5% n-propanol, which increases membrane permeability and causes mixing of storage and metabolic nitrate pools in the cells, was added to the assay medium lacking nitrate, anaerobic nitrite production increased and the difference caused by the precultivation disappeared. Higher K+ concentrations in unbalanced one-salt-solutions of KNO3 can cause higher membrane permeability by accentuating Ca2+ deficiency, which results in a faster penetration of NO3- from the storage pool to the sites of its reduction and in an easier penetration of NO2- out of the roots. Higher NH4+ concentrations can change nitrate compartmentation and diminish the metabolic NO3- pool which results in a slower nitrate reduction. Lower NH4+ concentrations in KNO3 solutions (15 mM NO3-) probably partially counteract the K+ dependent increase in membrane permeability. There is no simple, direct relationship between the so-called metabolic pool of nitrate (i.e., anaerobic nitrite production) and the level of nitrate reductase, but the velocity of nitrate reduction can be influenced by nitrate compartmentation in the cell.

L22 ANSWER 38 OF 66 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1953:26073 BIOSIS Full-text
DOCUMENT NUMBER: PREV19532700026169; BA27:26169
TITLE: Uber die Bildung von Zellulase bei pathogenen Mikroorganismen.
AUTHOR(S): AMMANN, A.

CORPORATE SOURCE: Eidg. Technischen Hochschule, Zurich
SOURCE: PHYTOPATH ZEITSCHR, (1952) Vol. 18, No. 4, pp. 416-446.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable
ENTRY DATE: Entered STN: May 2007
Last Updated on STN: May 2007

AB A method is described for determining by means of viscosity measurements, the 1,4-beta-glycoside compounds which cellulose yields under the action of cellulase in bacterial and fungal culture filtrates. Methyl cellulose, a water-soluble cellulose-ether, is used as the substrate for determining enzyme activity. All organisms tested produced cellulase when only glucose (or at least no cellulose) was available to them in the culture solution, as a source of carbohydrate. Exceptions were Mucor ramannianus, Escherichia coli, and Staphylococcus aureus which were unable to decompose cellulose at all. The lignin attacking, white rot organisms and Bacterium carotovorum, a protopectin decomposer, were outstanding in cellulase production under the nutritional conditions of this study. Temperature studies using Aspergillus niger and Penicillium luteum showed that the temperature range for mycelial growth coincides with that for cellulase production. ABSTRACT AUTHORS: S. B. Locke

L22 ANSWER 39 OF 66 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1989228047 EMBASE Full-text
TITLE: Antibacterial and immunostimulatory properties of chemotactic N-formyl peptide conjugates of ampicillin and amoxicillin.
AUTHOR: Bycroft, B.W.; Lockey, P.M.; Penrose, A.; Grout, R.J.; Williams, P.
CORPORATE SOURCE: Department of Pharmaceutical Sciences, The University, Nottingham NG7 2RD, United Kingdom.
SOURCE: Antimicrobial Agents and Chemotherapy, (1989) Vol. 33, No. 9, pp. 1516-1521.
ISSN: 0066-4804 CODEN: AMACCQ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 037 Drug Literature Index
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Dec 1991
Last Updated on STN: 12 Dec 1991

AB N-Formyl dipeptide conjugates of ampicillin and amoxicillin related to the chemotactic peptide N-formyl-L-methionyl-L-leucyl-L-phenylalanine were synthesized and assessed for antibacterial activity and affinity for the chemotactic peptide receptor of differentiated human promyelocytic leukemia (HL-60) cells. The conjugates and parent β-lactam antibiotics showed similar antibacterial activities against Escherichia coli and Staphylococcus aureus. The affinity of each conjugate for the chemotactic peptide receptor was determined in a competitive binding assay, using 3H-labeled N-formyl-L-methionyl-L-leucyl-L-phenylalanine. All conjugates bound to the receptor, but with affinities ranging from 1/3 to 1/100 that of the tritiated substrate. There was good correlation between receptor affinity and stimulation of chemotaxis. The peptide-antibiotic conjugates also stimulated the oxidative metabolism of the HL-60 cells by inducing the production of superoxide and hydrogen peroxide as determined by Luminol- and Lucigenin-enhanced chemiluminescence. These conjugates, based on N-formyl-L-methionyl-L-leucyl-

L-phenylalanine, thus combine both potent antibacterial and immunostimulatory properties within the same molecule.

L22 ANSWER 40 OF 66 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1978052135 EMBASE Full-text
TITLE: Hepatic microsomal N glucuronidation and nucleic acid binding of N hydroxy arylamines in relation to urinary bladder carcinogenesis.
AUTHOR: Kadlubar, F.F.; Miller, J.A.; Miller, E.C.
CORPORATE SOURCE: McArdle Lab. Cancer Res., Univ. Wisconsin Med. Cent., Madison, Wis. 53706, United States.
SOURCE: Cancer Research, (1977) Vol. 37, No. 3, pp. 805-814.
ISSN: 0008-5472 CODEN: CNREA8
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
023 Nuclear Medicine
028 Urology and Nephrology
029 Clinical and Experimental Biochemistry
037 Drug Literature Index
LANGUAGE: English
AB Uridine 5' diphosphoglucuronic acid fortified hepatic microsomes from dogs, rats, or humans rapidly metabolized [3H] N hydroxy 2 naphthylamine (N HO 2 NA) to a water soluble product that yielded 98% of the parent N hydroxy amine upon treatment with β glucuronidase. The metabolite was identified as N (β 1 glucosiduronyl) N hydroxy 2 naphthylamine from ultraviolet, infrared, and mass spectral analyses of the glucuronide and its nitrone derivative. Incubation of N hydroxy 1 naphthylamine (N HO 1 NA), N hydroxy 4 aminobiphenyl (N HO ABP), or the N hydroxy derivatives of 2 aminofluorene, 4 aminoazobenzene, or N acetyl 2 aminofluorene with uridine 5' diphosphoglucuronic acid fortified hepatic microsomes also yielded water soluble products. β Glucuronidase treatment released 80 to 90% of the [3H] N HO 1 NA and [3H] N HO ABP conjugates as tritiated ether extractable derivatives. N HO 1 NA, N HO 2 NA, and N HO ABP and the glucuronides of these N hydroxy arylamines were relatively stable and non reactive near neutral pH. At pH 5, the N glucuronide of N HO 2 NA and the presumed N glucuronides of N HO 1 NA and N HO ABP were rapidly hydrolyzed to the N hydroxy arylamines that were then converted to reactive derivatives capable of binding covalently to nucleic acids. These data support the concept that arylamine bladder carcinogens are N oxidized and N glucuronidated in the liver and that the N glucuronides are transported to the urinary bladder. The hydrolysis of the glucuronides to N hydroxy arylamines and the conversion of the latter derivatives to highly reactive electrophilic arylnitrenium ions in the normally acidic urine of dogs and humans may be critical reactions for tumor induction in the urinary bladder.

L22 ANSWER 41 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2009-R38180 [82] WPIDS
TITLE: New tetrahydro-1H-indole derivative useful for treating e.g. pain disorder, dermatological disease, metabolic disease, muscle disease, neurological disease, immunodeficiency disease, autoimmune disease, fibrosis and ophthalmic disease
DERWENT CLASS: B02
INVENTOR: ALBAUGH P A; CHOI H; CHOPIUK G; FAN Y; RUCKER P V; WANG Z
PATENT ASSIGNEE: (IRMI-N) IRM LLC
COUNTRY COUNT: 122

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2009140128	A2	20091119	(200982)*	EN	269[0]	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2009140128	A2	WO 2009-US43073	20090507

PRIORITY APPLN. INFO: US 2009-152872P 20090216
US 2008-52879P 20080513

AN 2009-R38180 [82] WPIDS

AB WO 2009140128 A2 UPAB: 20091222

NOVELTY - A tetrahydro-1H-indole derivatives (I), are new.

DETAILED DESCRIPTION - Tetrahydro-1H-indole derivatives of formula (I), their salts and solvates, are new. R1=group of formula (II);
X1-X4=CR9, C or N;
X5=C(R3)2, O, S, S(O)2, S(O) or NR3; m=1-4;
n=0-3;
R3=e.g. 1-8C alkyl, 6-14C aryl, 3-8C cycloalkyl, 2-14C heterocycloalkyl or 2-13C heteroaryl (all optionally mono to tri substituted), or
R3+R3=3-8C cycloalkyl fused with a heterocycle; R2=e.g. 6-14C aryl, 2-13C heteroaryl or 2-14C heterocycloalkyl (all optionally mono to tri substituted), and R9=H, 3-8C cycloalkyl or 1-8C alkyl, with specific provisos.

ACTIVITY - Cytostatic; Analgesic; Dermatological; Muscular-Gen.; Neuroprotective; Immunosuppressive; Osteopathic; Antiinflammatory; Ophthalmological; Antimicrobial; Respiratory-Gen.; Respiratory-Gen. Nephrotropic; Hepatotropic; Cardiovascular-Gen.; Vasotropic; Cardiant; Antiasthmatic; Antiulcer; Gastrointestinal-Gen.; Antipsoriatic; Antiallergic; Antiarthritic; Antirheumatic; Neuroprotective; Anti-HIV.

MECHANISM OF ACTION - Kinase insert domain receptor (KDR) inhibitor; Stimulating factor receptor (FMS); Macrophage colony-Stimulating Factor Receptor (c-FMS); Fms-like tyrosine kinase-3 (FLT3) inhibitor; c-Kit; Janus kinase 2 (JAK2) inhibitor; Janus kinase 3 (JAK3) inhibitor; Aurora; Platelet derived growth factor receptor (PDGFR) inhibitor; Lymphocyte-specific protein tyrosine kinase (LCK) inhibitor; Tyrosine kinase A (TrkA) inhibitor; Tyrosine kinase B (TrkB) inhibitor; Tyrosine kinase C (TrkC) inhibitor; IGF-IR; Anaplastic lymphoma kinase 4 (ALK4) inhibitor; ALK5; Anaplastic lymphoma Kinase inhibitor. The efficacy of 4-(5-(6-(2-(3-fluoro-phenyl)-pyrrolidin-1-yl)-imidazo(1,2- b)pyridazin-3-yl)-pyridin-2-yl)-piperazin-2-one (Ia) was evaluated for Tel-TrkB inhibitor activity using cell proliferation of Ba/F3 inhibiting assay, and IC50 value was measured. (Ia) Showed IC50 value of 0.0004 mu M.

USE - In a pharmaceutical composition useful for treating a kinase-mediated disease or condition in a patient. The disease or condition is cancer, proliferative diseases, pain disorder, dermatological disease, metabolic disease, muscle disease, neurodegenerative disease, neurological disease, immunodeficiency disease, immunologically-mediated disease, autoimmune disease, autoimmune mediated disease, bone disease, inflammatory disease, fibrosis, ophthalmic disease, infectious disease, viral disease, wound repair, respiratory disease, pulmonary disease, renal disease, kidney disease, liver disease, cardiovascular disease, vascular disease, heart disease, cell death and hyperplasiaan inflammatory disease; also for treating asthma, chronic obstructive pulmonary disease (COPD), adult respiratory distress syndrome (ARDS), ulcerative colitis, Crohns disease, bronchitis, dermatitis, allergic rhinitis, psoriasis, scleroderma, urticaria, rheumatoid arthritis, multiple

sclerosis, lymphoma, metastasis, anaplastic large-cell lymphoma, osteosarcoma, fibrosarcoma, melanoma, breast cancer, renal cancer, brain cancer, prostate cancer, colorectal cancer, thyroid cancer, ovarian cancer, pancreatic cancer, neuronal cancer, neuroblastoma, lung cancer, uterine cancer, gastrointestinal cancer, HIV or lupus; for treating a cell-proliferative condition. The cell-proliferative condition is lymphoma, metastasis, anaplastic large-cell lymphoma, osteosarcoma, fibrosarcoma, melanoma, breast cancer, renal cancer, brain cancer, prostate cancer, colorectal cancer, thyroid cancer, ovarian cancer, pancreatic cancer, neuronal cancer, neuroblastoma, lung cancer, uterine cancer or gastrointestinal cancer (preferably anaplastic large-cell lymphoma, pancreatic cancer, ovarian cancer or lung cancer) (all claimed).

L22 ANSWER 42 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2009-R36780 [79] WPIDS
TITLE: Use of 1-cyclopropyl-3-(3-(5-morpholin-4-ylmethyl-1H-benzoimidazol-2-yl)-1H-pyrazol-4-yl)-urea for treating e.g. disease state or condition mediated by kinase, pain, myocardial contraction, Paget's disease, liver fibrosis
DERWENT CLASS: B02
INVENTOR: CURRY J E; LYONS J F; RAWLINS D A; SQUIRES M S; THOMPSON N T; WALLIS N G
PATENT ASSIGNEE: (ASTE-N) ASTEX THERAPEUTICS LTD; (THOM-I) THOMPSON N T
COUNTRY COUNT: 122

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2009138799	A1	20091119	(200979)*	EN	216	[0]

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2009138799	A1	WO 2009-GB50520	20090514

PRIORITY APPLN. INFO: GB 2008-9774 20080530
GB 2008-8731 20080514
AN 2009-R36780 [79] WPIDS
AB WO 2009138799 A1 UPAB: 20091209
NOVELTY - In treatment of diseases mediated by kinase or its mutated form e.g. member of AXL family such as Axl, Mer and Sky (particularly Mer); prophylaxis or treatment of disease or condition e.g. pain, myocardial contraction, Paget's disease, liver fibrosis; and as antibacterial agent or neuroprotective agent; 1-cyclopropyl-3-(3-(5-morpholin-4-ylmethyl- 1H-benzoimidazol-2-yl)-1H-pyrazol-4-yl)-urea (I) or its salt is used.
DETAILED DESCRIPTION - In prophylaxis or treatment of disease state or condition mediated by kinase or its mutated form which is a member of AXL family such as Axl, Mer and Sky (particularly Mer), member of protein kinase C (PKC) family such as alpha , beta -I, beta -II, gamma , delta , epsilon , zeta, eta , theta , iota, lambda , and mu (particularly PKC- mu or PKC- gamma), member of colony-stimulating factor 1 (CSF-1)/platelet-derived growth factor (PDGF) receptor subfamily (particularly macrophage colony-stimulating factor 1 receptor (M-CSF- 1R or FMS)), member Mitogen- and stress-activated kinase (MSK) family such as MSK1 and MSK2 (particularly MSK2), member of rabbit death-associated protein (DAP) kinase-related apoptosis-inducing protein kinase family such as death receptor associatedkinase 1 and 2 (DRAK1 and DRAK2) (particularly DRAK1), salt-inducible kinase (SIK), member of 90kDa ribosomal S6 kinase (RSK) family such as RSK1-4 (particularly RSK2, RSK3, and

RSK4), member of p21 activated kinase (PAK) family (particularly PAK5), member of brain specific kinase family, brain specific kinases 1 and 2 (BRSK1/2) (particularly BrSK2), or member of Tousled-like kinase (TLK) family such as TLK1 or TLK2 (particularly TLK2); as antibacterial agent, neuroprotective agent, immunosuppressive agent or anti-osteolytic agent; and in the prophylaxis or treatment of disease or condition e.g. pain, coronary artery disease, myocardial contraction, Paget's disease, Coffin-Lowry syndrome, pancreatic adenocarcinoma, hepatosplenomegaly, hyperalgesia, 1-cyclopropyl-3-(3-(5-morpholin-4-ylmethyl-1H-benzoimidazol-2-yl)-1H- pyrazol-4-yl)-urea (I) or its salt, solvate or tautomer is used. ACTIVITY - Antibacterial; Cardiant; Vasotropic; Cardiovascular-Gen.; Hypotensive; Osteopathic; Nephrotropic; Gastrointestinal-Gen.; Cerebroprotective; Neuroleptic; Immunosuppressive; Antiinflammatory; Cytostatic; Analgesic; Dermatological; Auditory; Ophthalmological; Uropathic; Respiratory-Gen.; Neuroprotective; Antiarthritic; Antidiarrheic; Antirheumatic; Antiulcer; Vulnerary; Antitubercular; Tuberculostatic; Antileprotic.

MECHANISM OF ACTION - Protein kinase C (PKC)- mu inhibitor; Mer receptor tyrosine kinase inhibitor; Mitogen- and stress-activated kinase-2 (MSK2) inhibitor; Death receptor associated kinase 1 (DRAK1) inhobitor; p21 Activated kinase-5 (PAK5) inhibitor; Tousled-like kinase (TLK) inhobitor. The efficacy of the compound (I) was evaluated for the ribosomal S6 kinase 2 (RSK2) inhibitory activity using standard assay; and IC50 value was measured. The compound (I) showed IC50 value of 1-10 nM.

USE - As antibacterial agent, neuroprotective agent, immunosuppressive agent, and anti-osteolytic agent; and in the manufacture of medicament for prophylaxis or treatment of disease state or condition mediated by kinase; pain; coronary artery disease; myocardial contraction; cardiomyopathy e.g. dilated cardiomyopathy; cardiac remodeling; heart disease and its manifestations e.g. cardiac hypertrophy; heart failure e.g. congestive heart failure (CHF); hypertension e.g. chronic hypoxic pulmonary hypertension (PHTN) disorder; systemic vascular disease; lung conditions such as bronchiolitis e.g. tracheobronchitis, interstitial lung disease, and lung injury; disease state or condition resulting in excessive bone formation; Paget's disease; disease in which bone resorption mediates morbidity including prosthesis failure; osteolytic sarcoma; tumor metastasis to bone; osteolytic disease associated with bone metastasis; proliferative vitreoretinopathy; liver fibrosis; renal failure; irritable bowel syndrome (IBS); oxidative stress-related neurodegenerative disorder; diabetic nephropathy and neuropathy; cerebral ischemia; Coffin-Lowry syndrome; Borna disease; spinocerebellar ataxia type 14 (SCA14); schizophrenia; transplant rejection; organ transplantation; resistance to transplantation, graft versus host disease; pancreatitis; metal e.g. lead poisoning; pancreatic adenocarcinoma; gastric adenocarcinomas; invasive and/or metastatic breast cancer; metastasis from uterine, breast, prostate, lung, colon and stomach cancer; hairy cell leukemia particularly bone metastases; adenopathy e.g. lymphadenopathy; circulating lymphob lasts; allodynia including mechanical allodynia and ethyl alcohol or opiate withdrawal-associated allodynia; hyperalgesia particularly thermal hyperalgesia and hyperalgesia during ethyl alcohol or opiate withdrawal e.g. ethyl alcohol or opiate withdrawal-associated hyperalgesia; Gram-positive and Gram-negative bacterial infection; disease state or condition caused by S. aureus and P. aeruginosa; streptococcal infection selected from strep throat, impetigo, erysipelas, scarlet fever, infection from surgical procedures, hospital acquired lung infection, skin infection, diabetic foot infection, soft tissue infection, bone infection, joint infection, ear infection e.g. otitis media, eye infection e.g. conjunctivitis and blepharoconjunctivitis, urinary tract infection e.g. catheter infection, venous catheter insertions, prosthesis infection, respiratory tract infection e.g. upper respiratory tract infection, and lower respiratory tract infection; tonsillitis; meningitis; cellulitis; diverticulitis; endocarditis; osteomyelitis; pseudomembranous colitis; sinusitis; laryngitis; pneumonia e.g. community acquired pneumonia;

bronchopneumonia and legionellosis e.g. Legionnaires'disease; sepsis; septic arthritis; cellulitis; osteomyelitis; epiglottitis; exacerbation of existing chronic obstructive pulmonary disease (COPD); botulism; food poisoning; gonorrhoea; septicemia e.g. meningococcal septicemia; typhoid fever; paratyphoid fever; foodborne illness selected from food poisoning; toxic shock syndrome (TSS); gastrointestinal disease e.g. diarrhea, dysentery-like condition; ankylosing spondylitis; scalded skin syndrome; peptic ulcer; chronic gastritis; duodenitis; gas gangrene; enterotoxemia; tetanus; anthrax; listeriosis; necrotizing fascitis; tuberculosis; bacteremia; chancroid; shigellosis; and leprosy in mammals particularly human (all claimed). ADVANTAGE - The compound has improved solubility in aqueous solution, better physicochemical properties, improvement in drug metabolism and pharmacokinetic properties, improved stability i.e. shelf life and thermal stability, reduced dosage requirements; has improved potency versus therapeutic targets, cell activity in proliferation and clonogenic assays, anti-cancer activity and therapeutic index.

L22 ANSWER 43 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2009-N53907 [64] WPIDS
TITLE: Self-assembling peptide nanoparticle for vaccination comprises aggregates of building blocks formed of continuous chain of two peptide oligomerization domains linked by linker, where at least one domain is coiled-coil of T or B-cell epitope
DERWENT CLASS: B04; D16
INVENTOR: BURKHARD P
PATENT ASSIGNEE: (ALPH-N) ALPHA-O PEPTIDES AG
COUNTRY COUNT: 122

PATENT INFO ABBR.:

PATENT NO	KIND DATE	WEEK	LA	PG	MAIN IPC
WO 2009109428	A2 20090911	(200964)*	EN	93[8]	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2009109428	A2	WO 2009-EP50996	20090129

PRIORITY APPLN. INFO: EP 2008-101221 20080201
AN 2009-N53907 [64] WPIDS
AB WO 2009109428 A2 UPAB: 20091006
NOVELTY - A self-assembling peptide nanoparticle (P1) comprises: aggregates of a multitude of building blocks consisting of a continuous chain formed of two peptidic oligomerization domains linked by a linker, in which at least one of the peptidic oligomerization domains is/are coiled-coil that incorporates T- and/or B-cell epitope within the oligomerization domain, and the oligomerization domains and the linker are optionally further substituted. DETAILED DESCRIPTION - A self-assembling peptide nanoparticle (P1) comprises: aggregates of a multitude of building blocks of formula D1-L1-D2 (I) consisting of a continuous chain formed of two peptidic oligomerization domains linked by a linker, in which at least one of the peptidic oligomerization domains is/are coiled-coil that incorporates T- and/or B-cell epitope within the oligomerization domain, and the oligomerization domains and the linker are optionally further substituted. D1=a peptidic oligomerization domain having a tendency to form oligomers (D1)m;

D2=a peptidic oligomerization domain having a tendency to form oligomers (D2)n;
m and n=2-10;
L1=a bond or a short linker segment. Provided that:
(1) m is not equal n and not a multiple of n; and (2) n is not a multiple of m. INDEPENDENT CLAIMS are included for the following: (1) a composition (C1) comprising: the peptide nanoparticle (P1); and
(2) a monomeric building block of formula D1-L1-D2 (I). ACTIVITY - Antibacterial; Virucide; Antiparasitic; Cytostatic; Antiallergic; Antiaddicitve; Immunostimulant; Metabolic; Antismoking; Anti-HIV; Antimalarial; Amoebicide; Antipyretic; Antidiarrheic; Antiinflammatory; Neuroprotective; Respiratory-Gen.; Schistosomacide; Antitubercular; Tuberculostatic; Hepatotropic; Antihelmintic. No biological data given. MECHANISM OF ACTION - Vaccine.
USE - For forming a peptide nanoparticle composition useful for vaccinating a human or non-human animal comprising for inducing immune response against bacteria, viruses, parasites, cancer cells (where cancer is selected from brain, breast, liver, lung (non-small-cell lung, small-cell lung) cervical, colorectal, esophageal, ovarian, pancreatic, prostate, and renal cancers, glioblastoma, leukemia (acute myelogenous and chronic myeloid), lymphoma (non-Hodgkin's lymphoma), and melanoma); allergens, addictions, and diseases and metabolic disorders (preferably amebiasis, anthrax, Campylobacter infection, chickenpox, cholera, dengue, diphtheria, encephalitis, ebola, influenza (preferably Haemophilus influenzae Type b), Japanese encephalitis, leishmaniasis, malaria, measles, meningococcal disease, mumps, nosocomial infections, pertussis, pneumococcal disease, polio (poliomyelitis), rubella, shingles, schistosomiasis, tetanus, Tick-Borne encephalitis, trichomoniasis, trypanosomiasis, tuberculosis, typhoid, varicella, yellow fever, cytomegalovirus, Epstein-Barr Virus, foot and mouth disease virus (FMDV), Helicobacter pylori, hepatitis B virus, hepatitis C virus, hepatitis E virus, herpes simplex virus, human immunodeficiency virus, human papillomavirus, Neisseria meningitides, Pseudomonas aeruginosa, Staphylococcus aureus, Streptococcus pneumoniae, respiratory syncytial virus, rotavirus, roundworm, hookworm, and West Nile virus; and for inducing immune response in farm animals and/or a pet (claimed).
ADVANTAGE - The self-assembling peptide nanoparticles (SAPN) comprising aggregates of building blocks formed of continuous chain of two peptide oligomerization domains, in which at least one domain is coiled-coil of T and/or B-cell epitope. The oligomeric peptides form oligomers depending on the condition e.g. under denaturing conditions they are monomers, while under physiological conditions they form trimers. Under predefined conditions they adopt one single oligomerization state, which is needed for nanoparticle formation. The oligomerization state can be changed upon changing conditions, e.g. from dimers to trimers upon increasing salt concentration or from pentamers to monomers upon decreasing pH. The building block architecture is clearly distinct from viral capsid proteins that are composed of either one single protein, which forms oligomers of 60 or its multiple, or of more than one protein, which co-assemble to form the viral capsid structure. The SAPN nanoparticles are also clearly distinct from virus-like particles, as they are constructed from other than viral capsid proteins and that the cavity in the middle of the nanoparticle is too small to accommodate the DNA/RNA of a whole viral genome. The SAPN nanoparticles are formed from monomeric building blocks (I). If such building blocks assemble, they form even units. The monomeric building blocks assemble into such an even unit that is defined by the least common multiple (LCM). Hence, if the oligomerization domains of monomeric building block form a trimer ((D1)3) and pentamer ((D2)5), 15 monomers form even unit, which then assemble with linker in the form of a spherical peptidic nanoparticle. As the number of oligomerization domains D1 and D2 are not same or multiple of each other, the LCM is always larger than the number of respective oligomerization domains. The SAPN nanoparticles can also be formed

by the assembly of only at least one even units, which represent topologically closed structures.

L22 ANSWER 44 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2009-M56932 [55] WPIDS
TITLE: Treating, ameliorating, or preventing an autoimmune disorder, inflammatory disorder, transplant/graft rejection, lymphopenia, or graft-versus-host disease comprises administering an interleukin (IL)-21/IL-21R agonist
DERWENT CLASS: B04; D16; D21
INVENTOR: COLLINS M; LIU R; PIAO W; SHI F; VOLLMER T; YOUNG D A
PATENT ASSIGNEE: (AMHP-C) WYETH; (CATH-N) CATHOLIC HEALTHCARE WEST DBA ST MARYS ME
COUNTRY COUNT: 122

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2009100035	A2	20090813	(200955)*	EN	140	[17]

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2009100035	A2	WO 2009-US32894	20090202

PRIORITY APPLN. INFO: US 2008-25586P 20080201
AN 2009-M56932 [55] WPIDS
AB WO 2009100035 A2 UPAB: 20090826
NOVELTY - Treating, ameliorating, or preventing an autoimmune disorder, inflammatory disorder, transplant/graft rejection, lymphopenia, or graft-versus-host disease in a mammalian subject comprises administering to the subject an interleukin (IL)-21/IL-21R agonist, e.g. agonistic IL-21/IL-21R polynucleotides or fragments, agonistic IL-21/IL-21R polypeptides or fragments, agonistic anti-IL-21/IL-21R antibodies or fragments, and agonistic small molecules, in an amount sufficient to increase the level and/or activity of a Treg cell or a population of Treg cells in the mammalian subject.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are: (1) a method of transplanting/grafting an organ, tissue, cell, or group of cells to a mammalian subject comprising: (a) administering to the subject an IL-21/IL-21R agonist selected from agonistic IL-21/IL-21R polynucleotides or its fragments, agonistic IL-21/IL-21R polypeptides or its fragments, agonistic anti-IL-21/IL-21R antibodies or its fragments, and agonistic small molecules, in an amount sufficient to reduce the risk of transplant/graft rejection; and (b) transplanting/grafting the organ, tissue, cell, or group of cells to the subject, where the transplanting/grafting step (b) occurs either before, during, or after the administering (a);
(2) a method of treating, preventing, or ameliorating transplant/graft rejection in a mammalian transplant/graft recipient comprising: (a) detecting a symptom of transplant/graft rejection in a transplant/graft recipient; and (b) administering to the transplant/graft recipient an IL-21/IL-21R agonist selected from agonistic IL-21/IL-21R polynucleotides or its fragments, agonistic IL-21/IL-21R polypeptides or its fragments, agonistic anti-IL-21/IL-21R antibodies or its fragments, and agonistic small molecules;
(3) a method of treating, preventing, or ameliorating a cancer or an infectious disorder in a mammalian subject comprising administering to the subject an IL-21/IL-21R antagonist selected from antagonistic IL-21/IL-21R

polynucleotides or its fragments, antagonistic IL-21/IL-21R polypeptides or its fragments, antagonistic anti-IL-21/IL-21R antibodies or its fragments, and antagonistic small molecules, in an amount sufficient to decrease the level and/or activity of a Treg cell or a population of Treg cells in the mammalian subject; (4) a method of decreasing the transcription of HIV in a mammalian subject comprising administering to the subject an IL-21/IL-21R antagonist selected from antagonistic IL-21/IL-21R polynucleotides or its fragments, antagonistic IL-21/IL-21R polypeptides or its fragments, antagonistic anti-IL-21/IL-21R antibodies or its fragments, and antagonistic small molecules, in an amount sufficient to decrease the level and/or activity of Foxp3 in the mammalian subject;
(5) a method of modulating the level and/or activity of a Treg cell or a population of Treg cells in a mammalian subject comprising administering to the subject an IL-21/IL-21R agonist or an IL-21/IL-21R antagonist in an amount sufficient to modulate the level and/or activity of the Treg cell or population of Treg cells in the mammalian subject; (6) a method of modulating the level and/or activity of Foxp3 in a mammalian subject comprising administering to the subject an IL-21/IL-21R agonist or an IL-21/IL-21R antagonist in an amount sufficient to modulate the level and/or activity of Foxp3 in the mammalian subject; (7) a method for increasing the ability of a vaccine composition containing an antigen to elicit a protective immune response against the antigen in a mammalian subject comprising administering to the mammalian subject, either simultaneously with or sequentially to the vaccine composition, an effective adjuvanting amount of an IL-21/IL-21R antagonist, so the ability of the vaccine composition to elicit the protective immune response is increased; (8) a composition useful as a vaccine comprising an antigen from a pathogenic microorganism selected from a viral, bacterial, and parasitic microorganism, and an effective adjuvanting amount of an IL-21/IL-21R antagonist, in a carrier; and (9) a composition comprising a cancer cell or tumor cell antigen in combination with an effective adjuvanting amount of an IL-21/IL-21R antagonist, in a carrier.
ACTIVITY - Neuroprotective; Antiarthritic; Hemostatic; Antiinflammatory; Muscular-Gen; Hepatotropic; Dermatological; Immunomodulator; Immunosuppressive; Antidiabetic; Antipsoriatic; Endocrine-Gen; Antianemic; Antirheumatic; Gastrointestinal-Gen; Antiulcer; Antithyroid; Cytostatic; Antibacterial; Virucide; Antiparasitic; Immunostimulant. Test details are described but no results given.
MECHANISM OF ACTION - Vaccine; IL-21 Agonist; IL-21R Agonist.
USE - The methods are useful for treating, ameliorating, or preventing an autoimmune disorder, inflammatory disorder, transplant/graft rejection, lymphopenia, or graft-versus-host disease in a mammalian subject; transplanting/grafting an organ, tissue, cell, or group of cells to a mammalian subject; treating, preventing, or ameliorating transplant/graft rejection in a mammalian transplant/graft recipient; treating, preventing, or ameliorating a cancer or an infectious disorder; decreasing the transcription of HIV in a mammalian subject; modulating the level and/or activity of a Treg cell or a population of Treg cells in a mammalian subject; modulating the level and/or activity of Foxp3 in a mammalian subject; and increasing the ability of a vaccine composition containing an antigen to elicit a protective immune response against the antigen in a mammalian subject. The autoimmune disorder is multiple sclerosis, juvenile idiopathic arthritis, psoriatic arthritis, hepatitis C virus-associated mixed cryoglobulinemia, polymyositis, dermatomyositis, polyglandular syndrome type II, autoimmune liver disease, Kawasaki disease, myasthenia gravis, immunodysregulation polyendocrinopathy enteropathy X-linked (IPEX), type I diabetes, psoriasis, hypothyroidism, hemolytic anemia, thrombocytopenia, spondylarthritis, Sjogren's syndrome, rheumatoid arthritis, inflammatory bowel disease, Crohn's disease, ulcerative colitis, eczema, gastritis, and thyroiditis; the inflammatory disorder is contact hypersensitivity and atopic dermatitis; the cancer or infectious disorder is a cancer, and where the cancer is a solid tumor, a soft tissue

tumor, or a metastatic lesion, breast cancer, ovarian cancer, lung cancer, leukemia, lymphoma, melanoma, colorectal cancer, or renal cancer; the infectious disorder is caused by a bacterial, viral, or parasitic infection, e.g. caused by HIV infection, Schistosoma mansoni infection, hepatitis infection, Epstein-Barr virus infection, Borrelia infection, JC virus infection, cytomegalovirus infection, Coxsackie virus infection, papilloma virus infection, or herpes virus infection. It is also useful as a vaccine (all claimed).

L22 ANSWER 45 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2009-M53398 [57] WPIDS
TITLE: New crystalline or amorphous 17alpha-ethynyl-5alpha-androstane-3alpha,17beta-diol used for preparing medicament for treatment or prophylaxis of precancer e.g. prostatic intraepithelial neoplasia, or cancer e.g. prostate cancer
DERWENT CLASS: A96; B01
INVENTOR: LORIMER K R; OLSON E; STICKNEY D; WHITE S K; WOLFE B S
PATENT ASSIGNEE: (HOLL-N) HOLLIS-EDEN PHARM INC
COUNTRY COUNT: 122

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2009100258	A1	20090813	(200957)*	EN	124[26]	
US 20090291932	A1	20091126	(200978)	EN		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2009100258	A1	WO 2009-US33280	20090205
US 20090291932	A1 Provisional	US 2008-26472P	20080205
US 20090291932	A1 Provisional	US 2008-93694P	20080902
US 20090291932	A1 Cont of	WO 2009-US33280	20090205
US 20090291932	A1	US 2009-370510	20090212

PRIORITY APPLN. INFO: US 2008-93694P 20080902
US 2008-26472P 20080205
WO 2009-US33280 20090205
US 2009-370510 20090212

AN 2009-M53398 [57] WPIDS
AB WO 2009100258 A1 UPAB: 20090907
NOVELTY - Crystalline or amorphous 17 alpha -ethynyl-5 alpha -androstane-3 alpha ,17 beta -diol is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for: (1) a method of preparing a pharmaceutical formulation comprising contacting, mixing, and/or blending amorphous or crystalline 17 alpha -ethynyl-5 alpha -androstane-3 alpha ,17 beta -diol (1) with one, two, three, four, or more excipients to obtain a mixture; and processing the mixture to obtain pharmaceutical formulation; and (2) a method to treat a cancer or precancer in a subject comprising administering to the subject or delivering to the subject's tissues, an amount of crystalline compound (1), a formulation comprising crystalline compound (1) and excipient(s), or a formulation prepared from crystalline compound (1) and one, two, three, four, or more excipients.
ACTIVITY - Cytostatic; Keratolytic; Dermatological; Antiinflammatory; Antiallergic; Antipsoriatic. The effect of a formulation prepared with a polymorph of compound (1) (crystalline form III) on the rate of growth and

incidence of breast tumors induced by the administration of carcinogen N-methyl-nitrosourea (NMU) was determined. The activity of compound (1) was compared to TAXOTERE(RTM: docetaxel). Seven week old female Lewis rats (150 animals, 104 required for the study) were anesthetized with isoflurane for NMU administration. NMU was administered intraperitoneally at a dose of 50 mg/kg. Treatment commenced using a liquid formulation of compound (1) (4 mg/kg) prepared by dissolving crystalline form I in vehicle, when rats have a tumor volume of 0.5x 0.5 cm (at 12-20 week of age). Treatment continued for 28 consecutive days, followed by 28 days of observation. Mammary tumors were removed when a size of 2x 2 cm was reached in accordance with local institutional guidelines. Test result showed that compound (1) outperformed TAXOTERE(RTM: docetaxel) (p=0.042). Compound (1) treatment consistently showed less tumor burden than for the vehicle treated group (30% cyclodextrin-sulfobutylether in water) (day 7 on: p=less than 0.001).

MECHANISM OF ACTION - alpha 1-Adrenergic receptor antagonist; 5 alpha - Reductase inhibitor.

USE - The crystalline or amorphous compound (1) is useful for pharmaceutical formulation, which is a solid, optionally tablets, caplets, capsules, or another unit dosage form used for humans. It is used for preparing medicament for the treatment or prophylaxis of precancer, which is prostatic intraepithelial neoplasia (particularly high-grade prostatic intraepithelial neoplasia), cervical dysplasia, or ductal carcinoma in situ, or cancer, which is prostate cancer, breast cancer, ovarian cancer, endometrial cancer, lung cancer, pancreatic cancer, benign prostatic hypertrophy (all claimed), uterine cancer, adenocarcinoma, or malignant melanoma, in human or animal i.e. mammal or vertebrate such as non-human primate, dog, or rodent. It is useful for treating hyperproliferation conditions including hyperplasia (including endometrial hyperplasia, benign prostatic hyperplasia, and ductal hyperplasia), dysplasia, adenoma, sarcoma, blastoma, carcinoma, lymphoma, nevus, leukemia, papilloma, premalignant tumors, benign tumors, or malignant tumors including solid tumors and disseminated tumors such as one associated with or arising from prostate, lung, breast, ovary, skin, stomach, intestine, pancreas, neck, larynx, esophagus, throat, tongue, lip, oral cavity, oral mucosa, salivary gland, testes, liver, parotid, biliary tract, colon, rectum, cervix, uterus, vagina, pelvis, endometrium, kidney, bladder, central nervous system, glial cell, astrocyte, squamous cell, blood, bone marrow, muscle, or thyroid cells or tissue; and non-malignant hyperproliferative conditions of the skin including seborrheic keratosis, toxic eczema, allergic eczema, atopic dermatitis, ichthyosis, and psoriasis. Precancer also includes atypical small acinar proliferation.

ADVANTAGE - Compound (1) intermittent dosing can avoid or ameliorate some of the undesired aspects normally associated with discontinuous dosing, where undesired aspects include failure of the patient to adhere to a daily dosing regimen, and tendency to acquire disease tolerance to treatment or requirement to reduce the dosages of other therapeutic agents given concomitantly due to their associated unwanted side effects or toxicities.

L22 ANSWER 46 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2009-K60578 [43] WPIDS
TITLE: New 4-(4-phenyl-oxazol/thiazol-5-yl)-pyrimidine compounds useful for treating susceptible neoplasm e.g. breast cancer, melanomas, thyroid cancer, Barret's adenocarcinoma, cholangiocarcinoma, leukemias and lymphomas
DERWENT CLASS: B03; B04; C02; C03
INVENTOR: ADJABENG G; BIFULCO N; DAVIS-WARD R; DICKERSON S H; HORNBERGER K; PETROV K; RHEAULT T R; UEHLING D E; WATERSON A G
PATENT ASSIGNEE: (SMIK-C) SMITHKLINE BEECHAM CORP

COUNTRY COUNT: 122

PATENT INFO ABBR.:

PATENT NO	KIND DATE	WEEK	LA	PG	MAIN IPC
WO 2009076140	A1 20090618	(200943)*	EN	301[0]	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2009076140	A1	WO 2008-US85452	20081204

PRIORITY APPLN. INFO: US 2007-13409P 20071213

AN 2009-K60578 [43] WPIDS

AB WO 2009076140 A1 UPAB: 20090707

NOVELTY - 4-(4-Phenyl-oxazol/thiazol-5-yl)-pyrimidine compounds and their salts, are new.

DETAILED DESCRIPTION - 4-(4-Phenyl-oxazol/thiazol-5-yl)-pyrimidine compounds of formula (I) and their salts, are new. Y'=a moiety selected from moieties of formulae (i)-(iii); a=0-3;

R1, R2 and R4=e.g. halo, (halo)alkyl, -CN; Q1=-CH2- or -SO2-;

ring A1=cycloalkyl, phenyl, or 5-10 membered heteroaryl; b=0 or 1;

W'and W1=O, or S;

Q2=a bond, or -N(H)-;

R3=e.g. H, (halo)alkyl, alkenyl; ring B'=e.g. 5-6 membered heteroaryl, 9-10 membered heteroaryl, or phenyl;

e=0-3;

Z=e.g. halo, (halo)alkyl, alkenyl; c and d=0-2.

Full Definitions are given in the DEFINITIONS (Full Definitions) section.

INDEPENDENT CLAIMS are included for (1) preparation of the compound (I); and a pharmaceutical composition comprising: the compound (I), and a diluent or excipient. ACTIVITY - Cytostatic. N-(3-(2-Amino-5-(2-((3-((2-(dimethylamino)ethyl)oxy)-4-(methyloxy)phenyl)amino)-4-pyrimidinyl)-1,3-thiazol-4-yl)phenyl)-2,6-difluoro-N-methybenzamide (Id) was evaluated for inhibition of human head and neck tumor cells (HN5). In 96-well assays, cells (500) were plated in 105 mu l medium; next day compound (Id) diluted in dimethylsulfoxide (DMSO) and RPMI medium (270 mu l/well) with 10% fetal bovine serum (FBS) and 1% penicillin-streptomycin) were added to the plates. Final DMSO concentration was 0.2%. Total ATP was measured (as a surrogate estimate of cell number) using CellTiter-Glo (RTM: ATP measuring kit). % Inhibition of cell growth was calculated relative to DMSO vehicle-treated control wells, and IC50 was calculated and was found to be less than 100 nM.

MECHANISM OF ACTION - Receptor tyrosine kinase inhibitor; Serine/threonine kinase inhibitor; Tyrosine kinase inhibitor; v-Raf murine sarcoma viral oncogene (Raf) kinase inhibitor; Raf homolog b1 (B-Raf) inhibitor; Avian erythroblastic leukemia viral oncogene homolog B (ErbB) family kinase inhibitor; Epidermal growth factor receptor (EGFR) inhibitor; Erb-B2 inhibitor; Erb-B4 inhibitor. N-(3-(2-Amino-5-(2-((3-((2-(dimethylamino)ethyl)oxy)-4- (methyloxy)phenyl)amino)-4-pyrimidinyl)-1,3-thiazol-4-yl)phenyl)-2,6- difluoro-N-methylbenzamide (Id) was evaluated for ErbB-2 protein tyrosine kinase inhibition by substrate phosphorylation assay. In black 384-well polystyrene flat-bottom plate; substrate mix (containing 100 mM 3-(N-morpholino)propanesulfonic acid (MOPS) pH 7.5, 2 mM MnCl2, 20 mu M ATP, 0.01% Tween-20 (RTM: surfactant), 0.1 mg/ml bovine serum albumin (BSA), 0.8 mu M peptide substrate, and 1 mM dithiothreitol) (10 mu l); enzyme mix (10 mu l) (containing 100 mM MOPS, pH7.5; 0.01% Tween-20; 0.1 mg/ml BSA, and 10 nM ErbB2); and compound (Id) in dimethylsulfoxide (1 mu l, 0.00027-47.6 mu M) were incubated for 90 minutes at 20 degrees C, quenched by adding 100 mM EDTA

(20 mu l); and HTRF (RTM: cyclic adenosine monophosphate competitive immunoassay kit) detection mix (40 mu l) was added. The compound (Id) showed pIC50 against ErbB2 of greater than 7.
USE - For the preparation of a medicament for treating a susceptible neoplasm selected from bladder, breast, lung including small cell lung, non-small cell lung and squamous cell lung; cervical, colorectal, esophageal, gastric, ovarian, endometrial, pancreatic, prostate, renal, skin including melanomas; thyroid, and uterine cancers; Barret's adenocarcinoma, billiary tract carcinomas, cholangiocarcinoma, central nervous system tumors including primary CNS tumors and secondary CNS tumors, carcinoma of the head and neck, hematologic cancers including leukemias and lymphomas, hepatocellular carcinoma, pituitary adenoma, and sarcoma; in a mammal (e.g. human) (claimed).
The compounds are useful for inhibiting receptor tyrosine kinases and serine/threonine kinases including Raf family kinases (i.e. v-raf murine sarcoma viral oncogene homolog b1 (B-Raf)) and/or avian erythroblastic leukemia viral oncogene homolog B (ErbB) family kinases (i.e. epidermal growth factor receptor (EGFR), ErbB2 and ErbB4); and other multiple kinases such as insulin-like growth factor receptor (IGFR), insulin receptor (IR), insulin-related receptor (IRR), proto-oncogene tyrosine-protein kinase (Src), vascular endothelial growth factor receptor (VEGFR), platelet-derived growth factor receptor (PDGFR), mesenchymal epithelial transition factor (Met), v-yes-1 Yamaguchi sarcoma viral related oncogene homolog (Lyn), T cell-specific protein-tyrosine kinase (Lck), anaplastic lymphoma kinase 5 (Alk5), Aurora A and B, c-Jun-N-terminal kinase (JNK), tyrosine-protein kinase (Syk), mitogen activated protein kinase (p38), bruton agammaglobulinemia tyrosine kinase (BTK), focal adhesion kinase 1 (FAK), non-receptor tyrosine kinase Abelson oncogene (Abl), activated Cdc42-associated kinase 1 (Ack1), Abl-related Gene (Arg), B lymphocyte kinase (BLK), calmodulin-dependent protein kinase (CAMK)1-delta , cyclin dependent kinase 6 (CDK6), casein kinase 1 (CK1), c-kit hardy-zuckerman 4 feline sarcoma viral oncogene homolog (cKit), tyrosine protein kinase (CSK), discoidin domain receptor 2 (DDR2), Ephrin receptors, fibroblast growth factor receptor (FGFR), Fms-like tyrosine kinase (Flt3), Fms (receptor tyrosine kinase), proto-oncogene tyrosine-protein kinase (Fyn), hemopoietic cell kinase (Hck), homeodomain-interacting protein kinase 2 (HIPK2), interleukin-2 (IL-2)-inducible T cell kinase (Itk), misshapen/Nck intracellular kinase (NIK)s-related kinase (MINK), acetylmannosamine kinase 2 (Mnk2), P21-Activating Kinase 3 (PAK3), protein kinase C- mu (PKC mu), protein kinase D 2 (PKD2), protein-tyrosine kinase 5 (PTK5), ret proto-oncogene (Ret), Ron (receptor tyrosine kinase), Src-related intracellular tyrosine kinase (SIK), tyrosine kinase with immunoglobulin and egf factor homology domains 2 (Tie2), tyrosine kinase, receptor, type B (TrkB), and proto-oncogene tyrosine-protein kinase (Yes).
ADVANTAGE - The compounds are selective inhibitors of Raf family kinases or ErbB family kinases; such as by a factor of greater than or equal to 5; or selectively inhibit Raf family kinases and ErbB family kinases over other kinases i.e. are selective dual Raf/ErbB inhibitors. The compounds provide additive or potentially synergistic effects with some of the existing chemotherapies, radiation, biological or immunotherapeutics (including monoclonal antibodies) and vaccines; restore effectiveness of chemotherapies and radiation; and increase sensitivity of chemotherapies and/or radiation.

L22 ANSWER 47 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2009-E44407 [15] WPIDS
TITLE: New pyruvate compound in the form of ethyl pyruvate useful in preparation of oral supplement, e.g. energy drink, fruit juice for enhancing cell metabolism and absorption of medication in human or animal body
DERWENT CLASS: B06; D13

INVENTOR: ALEXANDER R; LORENZEN L H
PATENT ASSIGNEE: (ALEX-I) ALEXANDER R; (DONA-I) DONALD H J; (LORE-I) LORENZEN L H
COUNTRY COUNT: 122

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2009013723	A2	20090129	(200915)*	EN	19[0]	
WO 2009013723	A3	20090507	(200932)	EN		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2009013723	A2	WO 2008-IB52993	20080725
WO 2009013723	A3	WO 2008-IB52993	20080725

PRIORITY APPLN. INFO: US 2007-935123P 20070726
AN 2009-E44407 [15] WPIDS
AB WO 2009013723 A2 UPAB: 20090311
NOVELTY - Pyruvate compound in the form of ethyl pyruvate, is new.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included composition comprising pyruvate compound in the form of ethyl pyruvate and micro-clustered water.
ACTIVITY - Muscular-Gen.; Immunomodulator; Antidiabetic. The athletes was instructed to consume either CPR (RTM: terahertz treated water including ethyl pyruvate) (test) (0.5 l) or standard bottled water (control) (0.5 litre) 60 minutes prior to testing. The subjects again consumed test (0.5 l) or control (0.5 l) 30 minutes prior to testing. The results for VO2 (ml/kg) for each test/control subject, to measure the extraction and utilization of oxygen during the stationary bicycle test of these elite athletes, were calculated. The result for change (%) in extraction and utilization of oxygen for test/control was found to be 14.08/1.49.
MECHANISM OF ACTION - None given.
USE - For preparing oral supplement useful for enhancing cell metabolism and absorption of medications in a human or animal body. The oral supplement is useful in consumable beverage or food stuff selected from energy bar or diet bar, energy drink, soft drink, fruit juice, and coffee- or tea-based drinks (e.g. Camellia sinensis var sinensis and/or Camellia sinensis var assamica) (claimed). Also useful to lower body mass; promote enhanced cellular function of all cells dependant on mitochondrial generated ATP for optimal function, DNA self repair and normal cell life; decrease blood glucose level in diabetics; inhibit the production of cell and tissue damaging free radicals and aid as an effective antioxidant, helps to eliminate formed free radicals; improve cognitive, language, behavioral and social skills of autistic children and adults; promotes enhanced mental and physical recovery of cardiac, cancer and other chronically ill children and adults; and promotes enhanced energy recovery from mononucleosis, chronic fatigue syndrome, epstein barr, hepatitis viruses and other chronic debilitating conditions.
ADVANTAGE - The pyruvate compound increases overall energy and mental alertness, enhances muscle strength endurance, decreases fatigue, promotes mood enhancement and sense of well being; increases fat metabolism, fat loss, enhances the actions of other weight loss products, and decreases muscle degradation in HIV/aids and other muscle wasting and catabolic conditions. The compound is potable, easily ingestible form of ethyl pyruvate that will allow for the same cellular and organ benefits that are derived from intravenous ethyl pyruvate.

L22 ANSWER 48 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2008-J35710 [54] WPIDS
CROSS REFERENCE: 2008-C18853; 2008-D82706; 2008-D99602; 2008-F28995; 2008-G32646
DOC. NO. CPI: C2008-258382 [54]
DOC. NO. NON-CPI: N2008-672309 [54]
TITLE: Evaluating oncological characteristics of cells from tissue having cancerous, by implanting tumor tissue within matrix for measuring behavior, incubating tissue for growth of cells, measuring parameters to obtain data of behaviors
DERWENT CLASS: B04; B05; C03; C07; D16; S03; T01; W01
INVENTOR: COSTELLO P C; MCDONALD W B
PATENT ASSIGNEE: (COST-I) COSTELLO P C; (MCDO-I) MCDONALD W B
COUNTRY COUNT: 1

PATENT INFO ABBR.:

PATENT NO	KIND DATE	WEEK	LA	PG	MAIN IPC
US 20070292901	A1 20071220	(200854)*	EN	18[8]	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 20070292901	A1 Provisional	US 2006-813689P	20060615
US 20070292901	A1	US 2007-812127	20070615

PRIORITY APPLN. INFO: US 2007-812127 20070615
US 2006-813689P 20060615

AN 2008-J35710 [54] WPIDS
CR 2008-C18853; 2008-D82706; 2008-D99602; 2008-F28995; 2008-G32646
AB US 20070292901 A1 UPAB: 20080822
NOVELTY - Evaluating the oncological characteristics of cells from tissue suspected of being cancerous/pre-cancerous, by implanting sample quantity of suspected tumor tissue within a three-dimensional physiological matrix that adapted and arranged for measuring parameters of the behavior of cells of the suspected tumor tissue, providing nutrition to the cells so that the parameters can be measured, incubating tissue for the growth of tissue for a time sufficient to obtain measurements with respect to parameters, and measuring parameters to obtain data regarding behaviors.
DETAILED DESCRIPTION - Evaluating the oncological characteristics of cells from tissue suspected of being cancerous or pre-cancerous, involves obtaining a sample quantity of suspected tumor tissue or cells from the animal, where the tissue comprises one or more types of cells, implanting the sample quantity of suspected tumor tissue at least partially within a three-dimensional physiological matrix that adapted and arranged for measuring one or more parameters of the behavior of cells of the suspected tumor tissue, providing sufficient nutrition to the cells of tissue or cells so that the parameters can be measured, incubating or culturing the tissue and cells in an environment suitable for the growth of the cells and tissue for a time sufficient to obtain measurements with respect to parameters, and measuring parameters to obtain data regarding the behaviors.
USE - For evaluating the oncological characteristics of cells from tissue suspected of being cancerous or pre-cancerous in animals such as primates, humans, equines, bovines, canids, mammals, felines, canines, murines, porcines, avians, amphibians and reptilians (all claimed).
ADVANTAGE - The method permits the testing of many types of therapies including pharmaceutical compounds and non-chemical therapies. The methods

provide data useful for tailoring specific individual therapeutic regimens directed toward the precise tissue being evaluated.

L22 ANSWER 49 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2008-G32646 [40] WPIDS
CROSS REFERENCE: 2008-C18853; 2008-D82706; 2008-D99602; 2008-F28995; 2008-J35710
DOC. NO. CPI: C2008-201525 [40]
DOC. NO. NON-CPI: N2008-494531 [40]
TITLE: Evaluating therapeutic compound(s) with respect to tissue(s) from mammal, involves implanting sample of suspected tissue within three-dimensional physiological matrix, and incubating or culturing tissue and cells with therapeutic compounds
DERWENT CLASS: B04; D16; W01
INVENTOR: COSTELLO P C; MCDONALD W B
PATENT ASSIGNEE: (COST-I) COSTELLO P C; (MCDO-I) MCDONALD W B
COUNTRY COUNT: 1

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
US 20080070269	A1	20080320	(200840)*	EN	18[8]	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 20080070269	A1 Provisional	US 2006-813686P	20060615
US 20080070269	A1 Provisional	US 2006-813689P	20060615
US 20080070269	A1	US 2007-812128	20070615

PRIORITY APPLN. INFO: US 2007-812128 20070615
US 2006-813686P 20060615
US 2006-813689P 20060615

AN 2008-G32646 [40] WPIDS
CR 2008-C18853; 2008-D82706; 2008-D99602; 2008-F28995; 2008-J35710
AB US 20080070269 A1 UPAB: 20080624
NOVELTY - Evaluating therapeutic compound(s) with respect to tissue(s) from mammal involves implanting sample of suspected tissue within three-dimensional physiological matrix, incubating or culturing tissue and cells with therapeutic compounds in an environment for the growth of cells and tissue for a time to obtain measurements with respect to parameters, and measuring parameters to obtain data regarding behaviors with respect to therapeutic compounds.
DETAILED DESCRIPTION - Evaluating therapeutic compound(s) with respect to tissue(s) from a mammal comprises obtaining a sample of suspected tissue from mammal, implanting sample of suspected tissue at least partially within a three-dimensional physiological matrix, providing nutrition to cells and tissue so that parameters can be measured, providing therapeutic compound(s) to cells and tissue, incubating or culturing tissue and cells with therapeutic compounds in an environment for the growth of cells and tissue for a time to obtain measurements with respect to parameters, and measuring parameters to obtain data regarding behaviors with respect to therapeutic compounds. The tissue comprises cells and is suspected of being abnormal, cancerous or pre-cancerous. The matrix is arranged for measuring parameter(s) of the behavior of tissue and cells.

USE - The method is useful for evaluating therapeutic compound(s) with respect to tissue(s) from mammal, such as primates, humans, equines, bovines, canids, mammals, felines, canines, murines, porcines, avians, amphibians and reptilians. The method is also used for testing the efficacy of non-synthetic chemical therapies (claimed), particularly oncological testing.
ADVANTAGE - The method permits in vitro testing and analyses of conventional, experimental, or theoretical therapies with respect to specific target tissues or cells removed from a patient. The method uses sample portion that are not disrupted by trypsin or other enzymes but are mechanically divided into sample portions of sizes of testing. The method maintains the cell-to-cell contact of the sample tissue as if it were still in vivo. The method eliminates or reduces the problem of the prior art, i.e. lack of analytic tools. The method quickly obtain information useful for testing and evaluating specific therapies, e.g. therapeutic compounds to determine their effectiveness with respect to tumors or cancer types. The method saves time, effort and expense in terms of determination of appropriate therapeutic regimen.

L22 ANSWER 50 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2008-F28995 [35] WPIDS
CROSS REFERENCE: 2008-C18853; 2008-D82706; 2008-D99602; 2008-G32646; 2008-J35710
DOC. NO. CPI: C2008-172656 [35]
DOC. NO. NON-CPI: N2008-412964 [35]
TITLE: Evaluating efficacy of therapeutic compounds with respect to cancer cells, by obtaining sample from mammal, implanting sample within matrix, incubating or culturing sample with therapeutic compounds, and measuring parameters
DERWENT CLASS: B04; D16; W01
INVENTOR: COSTELLO P C; MCDONALD W B
PATENT ASSIGNEE: (COST-I) COSTELLO P C; (MCDO-I) MCDONALD W B
COUNTRY COUNT: 1

PATENT INFO ABBR.:

PATENT NO	KIND DATE	WEEK	LA	PG	MAIN IPC
US 20080070270	A1 20080320	(200835)*	EN	18[8]	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 20080070270	A1 Provisional	US 2006-813689P	20060615
US 20080070270	A1	US 2007-812152	20070615

PRIORITY APPLN. INFO: US 2007-812152 20070615
US 2006-813689P 20060615
AN 2008-F28995 [35] WPIDS
CR 2008-C18853; 2008-D82706; 2008-D99602; 2008-G32646; 2008-J35710
AB US 20080070270 A1 UPAB: 20080604
NOVELTY - Evaluating the efficacy of one or more therapeutic compounds with respect to tissue comprising cells, cancerous or pre-cancerous, by (a) obtaining a sample of the suspected tissue from the mammal, (b) implanting the sample within a three-dimensional matrix, (c) providing sufficient nutrition to the cells and tissue, (d) providing therapeutic compounds to the cells and tissue, (e) incubating or culturing the tissue and cells with therapeutic compounds, and (f) measuring the parameters to obtain data regarding the behaviors with respect to therapeutic compounds.

DETAILED DESCRIPTION - Method for evaluating the efficacy of one or more therapeutic compounds with respect to tissue from a mammal, the tissue comprising cells, the tissue being suspected of being abnormal, cancerous or pre-cancerous, involves (a) obtaining a sample of the suspected tissue from the mammal, where the tissue comprises one or more types of cells, (b) implanting the sample of the suspected tissue at least partially within a three-dimensional physiological matrix, the matrix being adapted and arranged for measuring one or more parameters of the behavior of the tissue and cells, (c) providing sufficient nutrition to the cells and tissue such that the parameters can be measured, (d) providing at least one or more therapeutic compounds to the cells and tissue, (e) incubating or culturing the tissue and cells with one or more therapeutic compounds in an environment suitable for the growth of the cells and tissue for a time sufficient to obtain measurements with respect to one or more parameters, and (f) measuring one or more of parameters to obtain data regarding the behaviors with respect to one or more therapeutic compounds. ACTIVITY - Cytostatic. No biological data given.

MECHANISM OF ACTION - None given.

USE - The method is useful for evaluating the efficacy of one or more therapeutic compounds with respect to tissue comprising cells, the tissue being suspected of being abnormal, cancerous or pre-cancerous, from a mammal (claimed).

ADVANTAGE - The method enables in vitro testing and analyses of one or more conventional, experimental or theoretical therapies with respect to specific target tissues or cells.

L22 ANSWER 51 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2008-D99602 [28] WPIDS
CROSS REFERENCE: 2008-C18853; 2008-D82706; 2008-F28995; 2008-G32646; 2008-J35710
DOC. NO. CPI: C2008-131561 [28]
DOC. NO. NON-CPI: N2008-312857 [28]
TITLE: Evaluating oncological characteristics of cells from animal tissue e.g. human, involves implanting sample of suspected tumor tissue in three-dimensional matrix to measure parameters behavior of cell; and providing nutrition to cells
DERWENT CLASS: B04; D16; S03
INVENTOR: COSTELLO P; MCDONALD W
PATENT ASSIGNEE: (COST-I) COSTELLO P; (MCDO-I) MCDONALD W
COUNTRY COUNT: 120

PATENT INFO ABBR.:

PATENT NO	KIND DATE	WEEK	LA	PG	MAIN IPC
WO 2008020337	A2 20080221	(200828)*	EN	41[8]	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2008020337	A2	WO 2007-IB3697	20070615

PRIORITY APPLN. INFO: US 1951-516171 20070613
US 2006-813686P 20060615
AN 2008-D99602 [28] WPIDS
CR 2008-C18853; 2008-D82706; 2008-F28995; 2008-G32646; 2008-J35710
AB WO 2008020337 A2 UPAB: 20080501

NOVELTY - Evaluating oncological characteristics of cells from tissue from an animal, involves implanting sample of suspected tumor tissue in a three-dimensional physiological matrix that is adapted and arranged for measuring parameters of behavior of cells of suspected tumor tissue; implanting sample in a three-dimensional physiological matrix that is adapted and arranged for measuring parameters of behavior of cells of suspected tumor tissue; providing nutrition to cells; incubating or culturing tissue and cells; and measuring parameters.

DETAILED DESCRIPTION - Evaluating oncological characteristics of cells from at least one tissue from an animal, where cells or tissue being suspected of being cancerous or precancerous, involves obtaining a sample quantity of suspected tumor tissue or cells from animal, where tissue comprises at least one types of cells; implanting the sample of the suspected tumor tissue at least partially within a three-dimensional physiological matrix that is adapted and arranged for measuring at least one parameters of the behavior of cells of suspected tumor tissue; providing sufficient nutrition to cells of tissue or cells so that parameters can be measured; incubating or culturing tissue and cells in an environment suitable for the growth of cells and tissue for a time sufficient to obtain measurements with respect to parameters; and measuring parameters to obtain data regarding behaviors. The sample is implanted with minimal disruption to cells or tissue. The method further involves transmitting data to evaluator. The tissue or cells are implanted in the matrix as the matrix is being formed; and providing at least one source of additional nutrition to tissue and cells so that parameters can be measured. The formed matrix comprises a gel or a permeable solid. The tissue or cells are implanted in the matrix after matrix is formed into a gel. The matrix is essentially free of bicarbonate when matrix is being formed into a gel. The matrix is formed in multiple containers. The physiological matrix has a pH which is adapted to a range suitable for particular tissue, a particular cell type or types, or to a particular tumor. The matrix comprises at least one natural or synthetic fiber. The physiological matrix is adapted and arranged for measuring the efficacy of at least one therapeutic compound. The physiological matrix is adapted and arranged for measuring the efficacy of at least one therapeutic compounds. The therapeutic compounds comprises at least two therapeutic compounds.

USE - For evaluating oncological characteristics of cells from tissue from animal e.g. primates, humans, equines, bovines, canids, mammals, felines, canines, murines, porcines, avians, amphibians and reptilians to test the efficacy of non-chemical therapies such as radiation therapies, brachio therapies, herbal therapies, naturopathic therapies, experimental therapeutic compounds, and new therapeutic compounds (claimed).

ADVANTAGE - The method permits the in vitro testing and analyses of conventional, experimental or theoretical therapies with respect to specific target tissues or cells. The method provides data for physicians and evaluator to make determination or estimate regarding the oncological status of a tissue and its cells. Thus from the data obtained efficient determination can be made regarding whether the tissue and cells are abnormal, precancerous or cancerous. The method permits the testing of many types of therapies, including pharmaceutical compounds and non-chemical therapies. The method makes it is possible to delay implantation for many hours or many days.

L22 ANSWER 52 OF 66	WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER:	2008-C18853 [16] WPIDS
CROSS REFERENCE:	2008-D82706; 2008-D99602; 2008-F28995; 2008-G32646; 2008-J35710
DOC. NO. CPI:	C2008-062859 [16]
DOC. NO. NON-CPI:	N2008-174977 [16]
TITLE:	Evaluating efficacy of compounds against cells in abnormal, cancerous or pre-cancerous tissue, involves

implanting tissue comprising cell types within three dimensional matrix, providing nutrients, incubating or culturing cells
DERWENT CLASS: B04; C06; D16; S03; W01
INVENTOR: COSTELLO P; MCDONALD W; NICKEL G; ROY A
PATENT ASSIGNEE: (COST-I) COSTELLO P; (MCDO-I) MCDONALD W; (NICK-I) NICKEL G; (ROYA-I) ROY A
COUNTRY COUNT: 120

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2008012696	A2	20080131	(200816)*	EN	42[8]	
US 20080026101	A1	20080131	(200881)	EN		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2008012696	A2	WO 2007-IB3494	20070615
US 20080026101	A1 Provisional	US 2006-813686P	20060615
US 20080026101	A1	US 2007-818509	20070614

PRIORITY APPLN. INFO: US 1951-415263 20070613
US 2006-813686P 20060615
US 2007-818509 20070614
AN 2008-C18853 [16] WPIDS
CR 2008-D82706; 2008-D99602; 2008-F28995; 2008-G32646; 2008-J35710
AB WO 2008012696 A2 UPAB: 20080306
NOVELTY - A method of evaluating the efficacy of compounds with respect to tissue from mammal, for activity against cells in abnormal, cancerous or pre-cancerous tissue, involves obtaining a tissue sample comprising one or more types of cells, implanting the tissue sample within a 3-dimensional physiological matrix, providing nutrients to the cells and incubating or culturing the cells in the presence of a candidate therapeutic compound.
DETAILED DESCRIPTION - A method for evaluating the efficacy of one or more therapeutic compounds with respect to at least one tissue from a mammal, where the tissue comprising cells, and the tissue being suspected of being abnormal, cancerous or pre-cancerous, involves (i) obtaining a sample of the suspected tissue from the mammal, where the tissue comprises one or more types of cells, (ii) implanting the sample quantity of the suspected tissue at least partially within a three-dimensional physiological matrix, the matrix being adapted and arranged for measuring one or more parameters of the behavior of the tissue and the cells, (iii) providing sufficient nutrition to the cells and the tissue so that the parameters can be measured, (iv) providing or subjecting at least one of the one or more therapeutic compounds or therapies to the cells and the tissue, (v) incubating or culturing the tissue and the cells with the one or more therapeutic compounds in an environment suitable for the growth of the cells and the tissue for a time sufficient to obtain measurements with respect to the one or more parameters, and (vi) measuring, with respect to each of the portions, one or more of the parameters to obtain data regarding the behaviors with respect to one or more therapeutic compounds.
USE - The method is useful for evaluating the efficacy of compounds with respect to tissue from mammal, for activity against cells in abnormal, cancerous or pre-cancerous tissue (claimed).
ADVANTAGE - The invention provides a rapid method to test one or more therapeutic substances to determine their efficacy against a particular tumor or type of tumor.

L22 ANSWER 53 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2008-B14744 [07] WPIDS
DOC. NO. CPI: C2008-031833 [07]
TITLE: New recombinant fungus that is oleaginous and produces a quinone-derived compound, useful for producing quinone-derived compounds, e.g. ubiquinones, for treating or preventing disorders, e.g. cardiovascular disorders or cancers
DERWENT CLASS: B05; D13; D16; D21
INVENTOR: BAILEY R B; MADDEN K T; TRUEHEART J; BAILEY R; MADDEN K
PATENT ASSIGNEE: (MICR-N) MICROBIA PRECISION ENG
COUNTRY COUNT: 119

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2007120423	A2	20071025	(200807)*	EN	541[2]	
WO 2007120423	A3	20080529	(200838)	EN		
EP 2004801	A2	20081224	(200903)	EN		
WO 2007120423	A9	20090423	(200929)	EN		
WO 2007120423	A8	20090528	(200935)	EN		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2007120423	A2	WO 2007-US6834	20070320
EP 2004801	A2	EP 2007-753460	20070320
EP 2004801	A2 PCT Application	WO 2007-US6834	20070320
WO 2007120423	A9	WO 2007-US6834	20070320
WO 2007120423	A8	WO 2007-US6834	20070320

FILING DETAILS:

PATENT NO	KIND		PATENT NO	
EP 2004801	A2	Based on	WO 2007120423	A

PRIORITY APPLN. INFO: US 2006-784499P 20060320
US 2006-848064P 20060928
AN 2008-B14744 [07] WPIDS
AB WO 2007120423 A2 UPAB: 20090509
NOVELTY - A recombinant fungus, where: (a) the recombinant fungus is oleaginous in that it can accumulate lipid to at least 20% of its dry cell weight; and (b) the recombinant fungus produces at least one quinone-derived compound selected from a ubiquinone, a vitamin K compound, and/or a vitamin E compound, and can accumulate the produced quinone-derived compound to at least 1% of its dry cell weight, is new.
DETAILED DESCRIPTION - A recombinant fungus, where: (a) the recombinant fungus is oleaginous in that it can accumulate lipid to at least 20% of its dry cell weight; and (b) the recombinant fungus produces at least one quinone-derived compound selected from a ubiquinone, a vitamin K compound, and/or a vitamin E compound, and can accumulate the produced quinone-derived compound to at least 1% of its dry cell weight; where the recombinant fungus comprises at least one modification as compared with a parental fungus, where the parental fungus both is not oleaginous and does not accumulate the quinone-derived compound to at least 1% of its dry cell weight, the modification is selected from quinonogenic modifications and/or oleaginic modifications; where the

modification alters oleaginicity of the recombinant fungus, confers to the recombinant fungus oleaginy, confers to the recombinant fungus the ability to produce the quinone-derived compound to a level at least 1% of its dry cell weight, or confers to the recombinant fungus the ability to produce at least one quinone-derived compound which the parental fungus does not produce, is new. INDEPENDENT CLAIMS are: (1) a strain of Y. lipolytica comprising one or more modifications selected from an oleaginic modification and/or a quinonogenic modification, so that the strain accumulates 1-15% of its dry cell weight as at least one quinone-derived compound; (2) a method of producing a quinone-derived compound; (3) an engineered Y. lipolytica strain that produces a ubiquinone contains one or more quinonogenic modifications; (4) an engineered S. cerevisiae strain comprising one or more quinonogenic modifications; (5) a method of preparing a food or feed additive or a dietary supplement containing a quinone-derived compound; (6) a composition comprising lipid bodies, at least one quinone-derived compound, and intact fungal cells; (7) a composition comprising an oil suspension comprising: lipid bodies, at least one quinone-derived compound, intact fungal cells, and a binder or filler; (8) a composition comprising an oil suspension comprising: lipid bodies, at least one quinone-derived compound, intact fungal cells, and one or more other agents selected from chelating agents, pigments, salts, surfactants, moisturizers, viscosity modifiers, thickeners, emollients, fragrances, and/or preservatives; (9) an isolated quinone-derived compound, prepared by a method comprising: (a) cultivating the fungus, the engineered Y. lipolytica strain, or the engineered S. cerevisiae strain under conditions that allow production of a quinone-derived compound; and (b) isolating the produced quinone-derived compound; (10) a composition comprising the quinone-derived compound further comprising one or more other agents; (11) a feedstuff comprising a quinone-derived compound in lipid bodies; (12) a quinone-derived compound composition comprising a Y. lipolytica cell containing at least 1% quinone-derived compound by weight; (13) a quinone-derived compound composition comprising Y. lipolytica lipid bodies, and at least one quinone-derived compound, where the quinone-derived compound is present at a level that is at least 1% by weight of the lipid bodies; and (14) a method comprising: (a) cultivating the fungus, the engineered Y. lipolytica strain, or the engineered S. cerevisiae strain under conditions that allow production of a quinone-derived compound; and (b) isolating the produced quinone-derived compound.

ACTIVITY - Cardiovascular-Gen; Cardiant; Hypotensive; Metabolic; Antidiabetic; Analgesic; Nootropic; Dermatological; Neuroprotective; Antiparkinsonian; Antiinflammatory; Cytostatic. No biological data given.

MECHANISM OF ACTION - None given.

USE - The recombinant fungus is useful for producing quinone-derived compounds, including ubiquinones, e.g. CoQ10 and/or C5-9 quinone compounds; vitamin K compounds, and vitamin E compounds. It can be used as food or feed additive; dietary or nutritional supplements; or compositions for nutraceutical, pharmaceutical and/or cosmetic use. The quinone-derived compounds can be used for treating or preventing disorders such as cardiovascular disorders (congestive heart failure, myocardial infarction, or hypertension), metabolic disorders, diabetes, pain, aging, neurodegenerative disorders (Parkinson's disease and Huntington's disease), inflammatory disorders, and cancers.

L22 ANSWER 54 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2007-526420 [51] WPIDS
DOC. NO. CPI: C2007-194313 [51]
TITLE: New substituted pyrazolo(4,3-c)pyridine derivatives useful as kinase inhibitors for treating e.g. cancer, viral infections, retinopathies, obesity, arthritis, and glomerulonephritis

DERWENT CLASS: B02
INVENTOR: ANGIOLINI M; BANDIERA T; LOMBARDI B A; LOMBARDI BORGIA A; NESI M; POLUCCI P; VARASI M; VILLA M; BORGIA A L
PATENT ASSIGNEE: (NERV-N) NERVIANO MEDICAL SCI SRL
COUNTRY COUNT: 118

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2007068619	A1	20070621	(200751)*	EN	238[1]	
EP 1968976	A1	20080917	(200862)	EN		
AU 2006326134	A1	20070621	(200864)	EN		
TW 200800437 7	A	20080116	(200908)	ZH		
US 20090023745	A1	20090122	(200908)	EN		
JP 2009518451	W	20090507	(200931)	JA	229	
CA 2631853	A1	20070621	(200932)	EN		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2007068619	A1	WO 2006-EP69285	20061206
AU 2006326134	A1	AU 2006-326134	20061206
EP 1968976	A1	EP 2006-841281	20061206
EP 1968976	A1 PCT Application	WO 2006-EP69285	20061206
US 20090023745	A1 PCT Application	WO 2006-EP69285	20061206
JP 2009518451	W PCT Application	WO 2006-EP69285	20061206
TW 200800437 7	A	TW 2006-145305	20061207
JP 2009518451	W	JP 2008-544955	20061206
US 20090023745	A1	US 2008-96979	20080617
CA 2631853	A1	CA 2006-2631853	20061206
CA 2631853	A1 PCT Application	WO 2006-EP69285	20061206
CA 2631853	A1 PCT Nat. Entry	CA 2006-2631853	20080603

FILING DETAILS:

PATENT NO	KIND		PATENT NO	
EP 1968976	A1	Based on	WO 2007068619	A
AU 2006326134	A1	Based on	WO 2007068619	A
JP 2009518451	W	Based on	WO 2007068619	A
CA 2631853	A1	Based on	WO 2007068619	A

PRIORITY APPLN. INFO: EP 2005-111959 20051212
AN 2007-526420 [51] WPIDS
AB WO 2007068619 A1 UPAB: 20090212
NOVELTY - Substituted pyrazolo(4,3-c)pyridine derivatives (I) are new.
DETAILED DESCRIPTION - Substituted pyrazolo(4,3-c)pyridine derivatives of formula (I) and their salts, solvates, N-oxides or prodrugs are new.
R=optionally substituted and optionally branched 1-6C alkyl, 3-6C cycloalkyl, heterocycloalkyl or aryl; R1=H, halo, nitro, NHCOR4, NHSO2R10, NR5R6, OR7, R8R9N-1-6C alkyl, R7O-1-6C alkyl or optionally substituted and optionally branched 1-6C alkyl;
R4=H, optionally substituted and optionally branched 1-6C alkyl, 3-6C cycloalkyl, heterocycloalkyl, aryl, NR8R9, R8R9N-1-6C alkyl or R7O-1-6C alkyl;
R5, R6=H, optionally substituted and optionally branched 1-6C alkyl, 3-6C cycloalkyl, heterocycloalkyl, aryl, R8R9N-2-6C alkyl or R7O-2-6C alkyl;
R7=H, optionally substituted and optionally branched 1-6C alkyl, 3-6C cycloalkyl, heterocycloalkyl, aryl and R8R9N-2-6C alkyl; R8, R9=H, optionally

substituted and optionally branched 1-6C alkyl, 3-6C cycloalkyl, heterocycloalkyl or aryl; or NR8R9=optionally substituted heterocycloalkyl; R10=optionally substituted and optionally branched 1-6C alkyl, 3-6C cycloalkyl, heterocycloalkyl or aryl; A, B, D, E=N, CH, CR2 or CR3; R2, R3=halo, trifluoromethyl, nitro, OR7, NR8R9, optionally substituted and optionally branched 1-6C alkyl, R8R9N-1-6C alkyl or R7O-1-6C alkyl; Ra, Rb=H or methyl; and
provided that:
(1) when Ra, Rb, and R1 are H, then at least one of A, B, D and E is N.
INDEPENDENT CLAIMS are also included for: (1) preparation of (I); (2) treating a disease caused by and/or associated with dysregulated protein kinase activity involves administrating (I); (3) inhibiting IGF1-R activity involving contacting the receptor with (I);
(4) a pharmaceutical composition comprising (I), and at least one excipient, carrier and/or diluent; and (5) a product or kit comprising (I) or the pharmaceutical compositions and at least one chemotherapeutic agent, as a combined preparation for simultaneous, separate or sequential use in anticancer therapy.
ACTIVITY - Cytostatic; Antidiabetic; Ophthalmological; Antiarteriosclerotic; Vasotropic; Immunosuppressive; Osteopathic; Antipsoriatic; Virucide; Respiratory-Gen.; Antiinflammatory; Antiarthritic; Nephrotropic.
MECHANISM OF ACTION - Kinase inhibitor. The efficacy of N-(5-(3,5-difluoro-benzenesulfonyl)-4,5,6,7- tetrahydro-1H-pyrazolo(4,3-c)pyridin-3-yl)-2-((S)-2-methoxy-1-methyl- ethylamino)-4-(4-methyl-piperazin-1-yl)-benzamide (Ia) was evaluated as IGF-1R inhibitor by trans-phosphorylation assay. (Ia) Showed IC50 value of 0.049 muM.
USE - For treating a disease caused by and/or associated with dysregulated protein kinase activity e.g. dysregulated IGF-1R or Aurora kinase activity such as cancer (e.g. carcinoma, squamous cell carcinoma, hematopoietic tumors of myeloid or lymphoid lineage, tumors of mesenchymal origin, tumors of the central and peripheral nervous system, melanoma, seminoma, teratocarcinoma, osteosarcoma, xeroderma pigmentosum, keratocanthomas, thyroid follicular cancer and Kaposi's sarcoma; breast cancer, lung cancer, colorectal cancer, prostate cancer, ovarian cancer, endometrial cancer, gastric cancer, clear cell renal cell carcinoma, uveal melanoma, multiple myeloma, rhabdomyosarcoma, Ewing's sarcoma), cell proliferative disorders, viral infections, retinopathies, diabetic and neonatal retinopathies and age related macular degeneration, atherosclerosis and conditions involving vascular smooth muscle proliferation or neointimal formation such as restenosis following angioplasty or surgery, graft vessel disease, such as can occur following vessel or organ transplantation, acromegaly and disorders secondary to acromegaly as well as other hypertrophic conditions in which IGF/IGF- IR signalling is implicated, such as benign prostatic hyperplasia, psoriasis, fibrotic lung disease, pulmonary fibrosis, pathologies related to chronic or acute oxidative stress or hyperoxia induced tissue damage, and metabolic disorders in which elevated IGF levels or IGF-IR activity are implicated, such as obesity; arthritis, glomerulonephritis and post-surgical stenosis and restenosis (claimed).
ADVANTAGE - (I) Are potent inhibitors of kinase enzymes. The method provides tumor angiogenesis and metastasis inhibition.

L22 ANSWER 55 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2007-343865 [32] WPIDS
DOC. NO. CPI: C2007-125322 [32]
TITLE: New pyrimidine derivatives used in production of an anti-proliferative effect and for the treatment of cancer
DERWENT CLASS: B02
INVENTOR: THOMAS A P; WOOD R; THOMAS A
PATENT ASSIGNEE: (ASTR-C) ASTRAZENECA AB; (ASTR-C) ASTRAZENECA UK LTD
COUNTRY COUNT: 116

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2007031745	A1	20070322	(200732)*	EN	130[0]	
EP 1931662	A1	20080618	(200841)	EN		
CN 101304996	A	20081112	(200906)	ZH		
JP 2009508833	W	20090305	(200917)	JA	111	
IN 2008DN01638	P1	20090320	(200951)	EN		
US 20090306116	A1	20091210	(200981)	EN		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2007031745 A1		WO 2006-GB3389	20060913
CN 101304996 A		CN 2006-80042247	20060913
EP 1931662 A1		EP 2006-779403	20060913
EP 1931662 A1	PCT Application	WO 2006-GB3389	20060913
CN 101304996 A	PCT Application	WO 2006-GB3389	20060913
JP 2009508833 W	PCT Application	WO 2006-GB3389	20060913
IN 2008DN01638 P1	PCT Application	WO 2006-GB3389	20060913
JP 2009508833 W		JP 2008-530610	20060913
IN 2008DN01638 P1		IN 2008-DN1638	20080225
US 20090306116 A1	Provisional	US 2005-717998P	20050916
US 20090306116 A1	PCT Application	WO 2006-GB3389	20060913
US 20090306116 A1		US 2008-66744	20080313

FILING DETAILS:

PATENT NO	KIND		PATENT NO	
EP 1931662	A1	Based on	WO 2007031745	A
CN 101304996	A	Based on	WO 2007031745	A
JP 2009508833	W	Based on	WO 2007031745	A

PRIORITY APPLN. INFO: US 2005-717998P 20050916
US 2008-66744 20080313

AN 2007-343865 [32] WPIDS

AB WO 2007031745 A1 UPAB: 20090130

NOVELTY - Pyrimidine derivatives and their salts are new.

DETAILED DESCRIPTION - Pyrimidine derivatives of formula (I) and their salts are new.

R1=cyano, 1-6C alkyl, amino, 1-4C alkylamino, di-(1-4C alkyl)amino, carbamoyl, 3-8C cycloalkyl, 3-8C cycloalkyl-1-6C alkyl or N(R1a)C(O)R1b; R1a,R1b=hydrogen or 1-6C alkyl (all optionally substituted by halogeno or 1-6C alkoxy);

q=0 - 3;

R2=H, halogeno or trifluoromethyl; R3=hydrogen, hydroxy, halogeno, 1-6C alkyl, 2-6C alkenyl, 2-6C alkynyl, 3-8C cycloalkyl, 3-8C cycloalkyl-1-6C alkyl, 1-6C alkoxy, 3-8C cycloalkyl-1-6C alkoxy, 1-6C alkylcarbonyl, 3-8C cycloalkylcarbonyl, 3-8C cycloalkyl-1-6C alkylcarbonyl, 1-6C alkoxycarbonyl, amino, 1-6C alkylamino, di-(1-6C alkyl)amino, 3-8C cycloalkylamino, 3-8C cycloalkyl-1-6C alkylamino, 1-6C alkoxyamino, carbamoyl, 1-6C alkylcarbamoyl, di-(1-6C alkyl)carbamoyl, C(O)R3b, OR3b, SR3b, NHR3b, N(1-6C alkyl)R3b, S(O)mR3a or N(R3c)C(O)R3a, saturated monocyclic 5- or 6-membered heterocyclic ring (comprising at least one ring heteroatom of N, O and S), 5- or 6-membered monocyclic heteroaromatic ring (comprising at least one ring heteroatom of N, O and S), phenyl, 2,7-diazaspiro(3.5)nonane group (all optionally substituted by 1-6C alkyl, 1-6C alkoxy, 1-6C alkoxy(1-6C)alkyl, 1-6C alkoxy-1-6C alkoxy,

halogeno, hydroxy, trifluoromethyl, tri-(1-4C alkyl)silyl, cyano, amino, 1-6C alkylamino, di-(1-6C alkyl)amino, 3-8C cycloalkylamino, 3-6C cycloalkyl-1-3C alkylamino, amino-1-6C alkyl, 1-6C alkylamino-1-6C alkyl, di-(1-6C alkyl)amino-1-6C alkyl, 3-8C cycloalkylamino-1-6C alkyl, 3-6C cycloalkyl-1-3C alkylamino(1-6C)alkyl, 1-6C alkoxycarbonyl, carbamoyl, 1-6C alkylcarbamoyl, di-(1-6C alkyl)carbamoyl, (1-6C)alkylthio, 1-6C alkylsulfonyl, 1-6C alkylsulfinyl, 1-6C alkylcarbonyl, alkanoylamino group of formula N(R3d)C(O)R3e, or a saturated monocyclic 3- - 7-membered ring (optionally comprising at least one heteroatom of N, O and S) and all optionally substituted 1-4C alkyl, hydroxy or cyano); m,n,p=0 - 2;
R3a=1-6C alkyl, 3-8C cycloalkyl, 3-8C cycloalkyl-1-6C alkyl or 1-6C alkoxy;
R3b=saturated monocyclic 4- - 6-membered heterocyclic ring comprising at least one ring heteroatom of N, O and S; R3c,R3d,R4-R15=hydrogen or 1-6C alkyl;
R3e=1-6C alkyl, 3-8C cycloalkyl, 3-8C cycloalkyl(1-6C)alkyl or 1-6C alkoxy group;
NQ1=a nitrogen-linked azetidinyl or pyrrolidinyl ring; Q2=a 5- or 6-membered monocyclic heteroaromatic ring (comprising at least one ring heteroatom of N, O and S and substituted by Q3 and is optionally substituted on any available ring atom by T1, halogeno, nitro, cyano, NR4R5, carboxy, hydroxy, 2-6C alkenyl, 3-8C cycloalkyl, 3-8C cycloalkyl-1-6C alkyl, 1-4C alkoxycarbonyl, 1-4C alkylcarbonyl, 2-6C alkanoylamino, phenylcarbonyl, S(O)p1-4C alkyl, C(O)NR6R7 or SO2NR8R9); R4+R5,R6+R7,R8+R9, R10+R11,R12+R13,R14+R15 (together with the nitrogen atom to which they are attached)=a saturated heterocyclic ring; T1=1-6C alkyl or 1-6C alkoxy (both optionally substituted by halogeno, amino, hydroxy and trifluoromethyl); Q3=1-6C alkyl, 3-8C cycloalkyl or 3-8C cycloalkyl(1-6C)alkyl group or optionally saturated 5- or 6-membered monocyclic ring (optionally comprising at least one ring heteroatom of N, O and S) and (all optionally substituted by T1, halogeno, nitro, cyano, NR10R11, carboxy, hydroxy, 2-6C alkenyl, 3-8C cycloalkyl, 1-6C alkoxycarbonyl, 1-6C alkylcarbonyl, 2-6C alkanoylamino, phenylcarbonyl, S(O)n(1-6C)alkyl, C(O)NR12R13 or SO2NR14R15;
CQ4=a carbon-linked 5- or 6-membered monocyclic heteroaromatic ring (comprising an imino group and at least one ring heteroatom of N, O and S in addition to the nitrogen atom of the imino group). The carbon atom linking CQ4 to the exocyclic NH group in the compound (I) is either the carbon atom of the imino group or, when present, a second ring carbon atom that is directly bonded to the nitrogen atom of the imino group. Any saturated monocyclic ring optionally bears 1 or 2 oxo or thioxo substituents. Provided that CQ4 is not pyrazole.
INDEPENDENT CLAIMS are included for the following: (1) a pharmaceutical product comprising the compound (I) or its salt and an additional anti-tumour agent for the conjoint treatment of cancer;
(2) preparation of the compound (I). ACTIVITY - Cytostatic.
MECHANISM OF ACTION - Inhibitor of insulin-like growth factor (IGF)-1 receptor (1R) activity. S-6-Methyl-4-(2-thiazolylamino)-2-(2-(3- pyrid-2-yl)isoxazol-5-yl)pyrrolidin-1-yl)pyrimidine (A1) was tested for inhibition of IGF-stimulated cell proliferation using murine fibroblasts (NIH3T3) over-expressing human IGF-1 receptor as given in WO 03/048133 and the compound (A1) showed IC50 value of 0.17 muM.
USE - In the manufacture of a medicament for use in the production of an anti-proliferative effect; in the treatment of a disease or medical condition mediated alone or in part by IGF-IR tyrosine kinase; in the prevention or treatment of those tumors which are sensitive to inhibition of IGF-IR tyrosine kinase involved in the signal transduction steps which lead to the proliferation of tumour cells in a warm-blooded anima; and for the prevention or treatment of those tumors which are sensitive to inhibition of IGF-IR tyrosine kinase involved in the signal transduction steps which lead to the proliferation of tumor cells in a warm-blooded animal and for the treatment of cancer (claimed).

ADVANTAGE - The compound exhibits potent anti-tumor activity and possesses potency against the IGF-1R tyrosine kinase.

L22 ANSWER 56 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2006-670264 [69] WPIDS
DOC. NO. CPI: C2006-205351 [69]
TITLE: New heterocyclic compounds are angiotensin converting enzyme inhibitors useful for treating cardiovascular disease such as heart failure, restenosis, hypertension and diastolic dysfunction
DERWENT CLASS: B02; B03
INVENTOR: GARVEY D S; GARVEY D
PATENT ASSIGNEE: (NITR-N) NITROMED INC
COUNTRY COUNT: 112

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2006102071	A1	20060928	(200669)*	EN	108[0]	
AU 2006227439	A1	20060928	(200801)	EN		
EP 1877374	A1	20080116	(200807)	EN		
JP 2008533171	W	20080821	(200857)	JA	95	
US 20080293678	A1	20081127	(200881)	EN		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2006102071	A1	WO 2006-US9698	20060317
AU 2006227439	A1	AU 2006-227439	20060317
EP 1877374	A1	EP 2006-738726	20060317
JP 2008533171	W	JP 2008-502091	20060317
US 20080293678	A1 Provisional	US 2005-662931P	20050318
EP 1877374	A1 PCT Application	WO 2006-US9698	20060317
JP 2008533171	W PCT Application	WO 2006-US9698	20060317
US 20080293678	A1 PCT Application	WO 2006-US9698	20060317
US 20080293678	A1	US 2008-886461	20080602

FILING DETAILS:

PATENT NO	KIND		PATENT NO	
AU 2006227439	A1	Based on	WO 2006102071	A
EP 1877374	A1	Based on	WO 2006102071	A
JP 2008533171	W	Based on	WO 2006102071	A

PRIORITY APPLN. INFO: US 2005-662931P 20050318
US 2008-886461 20080602

AN 2006-670264 [69] WPIDS

AB WO 2006102071 A1 UPAB: 20061027
NOVELTY - Heterocyclic compounds (I)-(III) are new.
DETAILED DESCRIPTION - Heterocyclic compounds of formulae (I) - (III) are new.
X6=R22 or -NHCH(C(O)OH.Z)(CH2C6H5); Y6=CH2SR21, -P(O)(OH.Z)((CH2)4C6H5), -OP(O)(O(CH(CH(CH3)2)(OC(O)CH2CH3))(CH2)4C6H5), -NHCH(C(O)R22)(CH2CH2C6H5) or NHCH(C(O)R22)(CH2CH2CH3);
W6=CH2, N(CH3), (1,3)dithiolane-2,2-diyl or =CH(cyclohexyl); Z7=H, methyl or (CH2)4-NH2; R19 and R20=H; or
R19+R20=oxo; or

R20+W6=-(CH2)4- or (CH2)3-;
R21=C(O)CH2CH3, H or moiety of formula (Ia); R22=OCH2CH3 or OH.Z;
Z=organic base or -N(R38)(R39)(R40); R38 - R40=K or Re; or
R38R39N=heterocycle;
L and K=(W3)a-Eb -(C(Re)(Rf))p1-Ec -(C(Re)(Rf))x-(W3)d-(C(Re)(Rf))y-(W3)i-Ej-(W3)g-(C(Re)(Rf)z-V4; a - d, g, i and j=0 - 3;
p1, x - z=0 - 10;
V4=V3, Re, -U3-V5 or V6;
V3=group of formula (Ib), (If)-(Ij) or (Im)-(Ip); R24=C6 H4 R27, -CN, S(O)2-C6 H4 R27, C(O)-N(Ra)(Ri), NO2 or -C(O)-OR25;
R25=aryl, lower alkyl, haloalkyl, hydroxyalkyl or arylalkyl; R26=C(O) or S(O)2;
R27=H, CN, S(O)2R25, C(O)N(Ra)(Ri), NO2 or -C(O)-OR25; T'=O, S or NR6;
R6=H, lower alkyl or aryl;
V6=group of formulae (Ic), (Id), (Iq) or (Ir); Z5=CH2 or O;
Z6=CH or N;
W3=C(O), C(S), T3, (C(R6)(Rf))h -, -N(Ra)Ri, alkyl, aryl, heterocycle, arylheterocyclic ring, (CH2 CH2 O) q 1 or a heterocyclic nitric oxide donor;
E=-T3-, alkyl, aryl, C(Re)(Rf)h-, heterocycle, arylheterocyclic ring, (CH2 CH2 O)q1 or Y3;
Y3=group of formulae (Ie), (Is)-(Iw); T=S(O)o, carbonyl or covalent bond; o=0 - 2;
Rj and Rk=alkyl or aryl; or
RjRkN=heterocycle;
T3=covalent bond, carbonyl, O, S(O)o or N(Ra)Ri; h=1 - 10;
q1=1 - 5;
Re and Rf=S1, (C(Ro)(Rp))k1-U3 -V5, -(C(Ro)(Rp))k1-U3-V3, (C(Ro)(Rp))k1-U3-V6 or -(C(Ro)(Rp))k1-U3-C(O)-V6; S1=H, alkyl, cycloalkoxy, halo, hydroxy, hydroxyalkyl, alkoxyalkyl, arylheterocyclic ring, alkylaryl, alkylcycloalkyl, alkylheterocyclic ring, cycloalkylalkyl, cycloalkylthio, arylalkylthio, arylalkylthioalkyl, alkylthioalkyl, cycloalkenyl, heterocyclicalkyl, alkoxy, haloalkoxy, amino, alkylamino, dialkylamino, arylamino, diarylamino, alkylarylamino, alkoxyhaloalkyl, sulfonic ester, alkylsulfonic acid, (aryl)sulfonic acid, arylalkoxy, alkylthio, arylthio, CN, aminoalkyl, aminoaryl, aryl, arylalkyl, alkylaryl, carboxamido, alkylcarboxamido, arylcarboxamido, amidyl, carboxyl, carbamoyl, alkylcarboxylic acid, arylcarboxylic acid, alkylcarbonyl, arylcarbonyl, ester, carboxylic ester, alkylcarboxylic ester, arylcarboxylic ester, sulfonamido, alkylsulfonamido, arylsulfonamido, alkylsulfonyl, alkylsulfonyloxy, arylsulfonyl, arylsulfonyloxy, sulfonic ester, alkyl ester, aryl ester, urea, phosphoryl, nitro, -U3-V5 or V6; or ReRfC and RoRpC=carbonyl, methanthial, heterocycle, cycloalkyl, aryl, oxime, imine, hydrazone, bridged cycloalkyl or formulae (IIa) or (IIb);
Ro and Rp=S1;
U3=O, S or N(Ra)Ri;
V5=NO or NO2;
k1=1 - 3;
Ra=lone pair of electrons, H or alkyl; Ri=H, alkyl, aryl, alkylcarboxylic acid, arylcarboxylic acid, alkylcarboxylic ester, arylcarboxylic ester, alkylcarboxamido, arylcarboxamido, alkylaryl, alkylsulfinyl, alkylsulfonyl, alkylsulfonyloxy, arylsulfinyl, arylsulfonyl, arylsulfonyloxy, sulfonamido, carboxamido, carboxylic ester, aminoalkyl, aminoaryl, -CH2-C-(U3-V5)(Re)(Rf), double bond to adjacent atom or -(N2O2-).M1+;
M1+=(in)organic cation;
B6=CH2, N;
G6=CH2 or S;
D6=CH2 or =CH(thiophen-2-yl); or B6+D6=phenyl;
Q6=H;
X7=H;
Y7=indane-2-yl; or

X7+Y7=formula (IIIa) or -C(O)N(CH3)CH2-; and R23=H or OCH3.
When B6 is a nitrogen and Q6 is CH2, B6+Q6 is hexahydropyridazine. Provided that:
(1) when R38 and R39 form an aromatic heterocycle it can be substituted at any position by L and R39 is not present; and (2) (I) must contain at least one organic nitric oxide enhancing compound linked via a salt bridge to at least one carboxylic and/or phosphinic acid group in (I).
INDEPENDENT CLAIMS are also included for the following: (1) a composition comprising (I), (II) or (III) and a carrier; and (2) a kit comprising at least one compound (I), (II) or (III)). ACTIVITY - Cardiovascular-Gen.; Cardiant; Vasotropic; Antianginal; Antiarteriosclerotic; Anticoagulant; Thrombolytic; Antilipemic; Cerebroprotective; Antiarrhythmic; Hepatotropic; Gynecological; Osteopathic; Nephrotropic; Ophthalmological; Antilipemic; Hypotensive.
MECHANISM OF ACTION - Angiotensin converting enzyme inhibitor.
USE - For treating a cardiovascular disease e.g. heart failure, restenosis, hypertension, diastolic dysfunction, coronary artery disease, myocardial infarction, postmyocardial infarction, cerebral infarction, arterial stiffness, atherosclerosis, atherogenesis, cerebrovascular disease, angina, aneurysm, ischemic heart disease, cerebral ischemia, myocardial ischemia, thrombosis, platelet aggregation, platelet adhesion or smooth muscle cell proliferation; a vascular or non-vascular complication associated with the use of a medical device; a wound associated with the use of a medical device; vascular or non-vascular wall damage; peripheral vascular disease; neointimal hyperplasia following percutaneous transluminal coronary angiography; vascular grafting; coronary artery bypass surgery; thromboembolic events; post-angioplasty restenosis, coronary plaque inflammation; hypercholesterolemia; embolism; stroke; shock; arrhythmia; atrial fibrillation or atrial flutter; thrombotic occlusion and reocclusion cerebrovascular incidents; left ventricular dysfunction and hypertrophy; renovascular disease e.g. renal failure, renal insufficiency, renal deterioration associated with severe hypertension or renovascular hypertension; diabetes; oxidative stress; endothelial dysfunctions; disease caused by endothelial dysfunctions; cirrhosis; pre-eclampsia; osteoporosis; nephropathy; peripheral vascular disease; portal hypertension; ophthalmic disorder; metabolic syndrome; hyperlipidemia (all claimed).
ADVANTAGE - The compounds are excellent angiotensin converting enzyme inhibitors.

L22 ANSWER 57 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2006-413370 [42] WPIDS
DOC. NO. CPI: C2006-130234 [42]
DOC. NO. NON-CPI: N2006-342340 [42]
TITLE: Medical device, useful to prevent and/or treat restenosis, in-stent restenosis and/or thrombosis, comprises medical implant component having metal surface (bound to chemical entity via chelator, or bound to chelator)
DERWENT CLASS: A96; B04; B05; D22; P32; P34
INVENTOR: GENGRINOVITCH S
PATENT ASSIGNEE: (GENG-I) GENGRINOVITCH S; (NOVI-I) NOVIK S; (STEN-N) STENTOMICS INC
COUNTRY COUNT: 112

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
US 20060115514	A1	20060601	(200642)*	EN	71[4]	
WO 2006056984	A2	20060601	(200642)	EN		

EP 1827528 A2 20070905 (200760) EN
IN 2007KN02327 P2 20070817 (200780) EN
AU 2005308452 A1 20060601 (200818) EN
KR 2007095916 A 20071001 (200819) KO
CN 101111273 A 20080123 (200833) ZH
JP 2008521476 W 20080626 (200844) JA 90

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 20060115514 A1	Provisional	US 2004-630560P	20041126
US 20060115514 A1		US 2005-284832	20051123
AU 2005308452 A1		AU 2005-308452	20051123
CN 101111273 A		CN 2005-80047303	20051123
EP 1827528 A2		EP 2005-804715	20051123
WO 2006056984 A2		WO 2005-IL1247	20051123
EP 1827528 A2		WO 2005-IL1247	20051123
IN 2007KN02327 P2		WO 2005-IL1247	20051123
KR 2007095916 A		WO 2005-IL1247	20051123
CN 101111273 A		WO 2005-IL1247	20051123
IN 2007KN02327 P2		IN 2007-KN2327	20070622
KR 2007095916 A		KR 2007-714549	20070626
JP 2008521476 W		WO 2005-IL1247	20051123
JP 2008521476 W		JP 2007-542511	20051123

FILING DETAILS:

PATENT NO	KIND		PATENT NO	
EP 1827528	A2	Based on	WO 2006056984	A
AU 2005308452	A1	Based on	WO 2006056984	A
KR 2007095916	A	Based on	WO 2006056984	A
CN 101111273	A	Based on	WO 2006056984	A
JP 2008521476	W	Based on	WO 2006056984	A

PRIORITY APPLN. INFO: US 2005-284832 20051123
US 2004-630560P 20041126
AN 2006-413370 [42] WPIDS
AB US 20060115514 A1 UPAB: 20090918
NOVELTY - Medical device (A) comprises a medical implant component having a metal surface (M) to which is bound a chemical entity (X) via a chelator (C) chelated to the metal surface, or comprises a medical implant component having (M) to which (C) is chelated.
DETAILED DESCRIPTION - Medical device (A) comprises a medical implant component having a metal surface (M) to which is bound a chemical entity (X) via a chelator (C) chelated to the metal surface in an (M)-(C)-(X) configuration, or comprises a medical implant component having (M) to which (C) is chelated.
INDEPENDENT CLAIMS are also included for: (1) a method of manufacturing (A); (2) a method of implanting (A) comprising implanting (A) in a subject; and (3) a method of preventing and/or treating a medical condition of a subject, comprising implanting (A) in the subject, such that activity of the bound chemical entity exhibits an efficacy for preventing and/or treating the medical condition; and (4) a chelate type of coordination compound of (C)-(Y).
(C) = a chelator; and
(Y) = a chemical entity (a drug chelated to the chelator or a biological moiety chelated to the chelator or a linker having a first part chelated to the chelator and having a second part bonded to a drug or a biological moiety).

ACTIVITY - Cardiovascular-Gen.; Vasotropic; Anticoagulant; Thrombolytic; Antiarrhythmic; Anorectic; Uropathic; Cytostatic; Antiinflammatory; Immunosuppressive; Vulnerary; Antilipemic; Antianemic; Diuretic.
MECHANISM OF ACTION - Alpha-adrenergic blocker; Angiotensin converting enzyme inhibitor; Beta-adrenergic blocker; Calcium channel blocker; Peripheral adrenergic antagonist; Extracellular matrix modulator.
USE - (A) is useful to prevent and/or treat a cardiovascular type of medical condition (restenosis, in-stent restenosis and/or thrombosis) of a subject, where the drug is alpha-adrenergic blocking drugs, angiotensin converting enzyme inhibitor drugs, antiarrhythmic drugs, anticoagulant and antiplatelet drugs, antithrombotic or thrombin inhibitor drugs, beta adrenergic blocking drugs, calcium channel blocking drugs, centrally acting drugs, cholesterol lowering agent drugs, digitalis drugs, diuretic drugs, nitrate drugs, peripheral adrenergic antagonist drugs and/or vasodilator drugs, or anti-neoplastic or anti-inflammatory drugs, immunosupressive or anti-proliferative drugs, migration inhibitor or extracellular matrix modulator drugs, or enhanced healing or re-endothelialization drugs (claimed). No biological data given.
ADVANTAGE - (A) provides a sufficiently effective, consistent, robust, and safe, solution to restenosis, and in-stent restenosis.

L22 ANSWER 58 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2006-381570 [39] WPIDS
CROSS REFERENCE: 2007-761198
DOC. NO. CPI: C2006-122929 [39]
TITLE: Composition, useful for analysis of chemical compound e.g. drug, to identify its activity profile in cell, comprises panel of assays, performed in cell, involving measurement of at least one molecular parameter
DERWENT CLASS: B04; D16
INVENTOR: KEON B; LAMERDIN J; MACDONALD M L; MICHNICK S W; WESTWICK J K; MACDONALD M; MICHNICK S; WESTWICK J
PATENT ASSIGNEE: (ODYS-N) ODYSSEY THERA INC
COUNTRY COUNT: 112

PATENT INFO ABBR.:

PATENT NO	KIND DATE	WEEK	LA	PG	MAIN IPC
WO 2006058014	A2 20060601	(200639)*	EN	115[24]	
US 20060160109	A1 20060720	(200648)	EN		
EP 1836631	A2 20070926	(200763)	EN		
AU 2005309649	A1 20060601	(200765)	EN		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2006058014	A2	WO 2005-US42344	20051122
US 20060160109	A1 Provisional	US 2004-629558P	20041122
US 20060160109	A1	US 2005-282745	20051121
EP 1836631	A2	EP 2005-824951	20051122
EP 1836631	A2	WO 2005-US42344	20051122
AU 2005309649	A1	AU 2005-309649	20051122

FILING DETAILS:

PATENT NO	KIND	PATENT NO

EP 1836631 A2 Based on WO 2006058014 A
AU 2005309649 A1 Based on WO 2006058014 A

PRIORITY APPLN. INFO: US 2005-282745 20051121
US 2004-629558P 20041122

AN 2006-381570 [39] WPIDS
CR 2007-761198
AB WO 2006058014 A2 UPAB: 20060620
NOVELTY - A composition (C1) comprising panel of assays, where each assay of the panel is performed in cell(s) and comprising a measurement of at least one molecular parameter, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) analysis (m1) of a test chemical compound to identify an activity profile of the compound in cell(s), comprising: (a) constructing (C1);
(b) contacting each of the cell(s) in the panel with a test chemical compound;
(c) measuring the effects of the compound in the assays in the panel; and
(d) using the results to identify an activity profile for the chemical compound in the cells;
(2) determining (M2) a profile of activity of a test compound in cell(s), comprising:
(a) constructing a panel of assays, comprising at least a first cell-based assay and a second cell-based assay, where each of the assay comprises a measurement of at least one molecular parameter; (b) contacting the first of two identical populations of cells from the first cell-based assay with a test chemical compound; (c) contacting the second of two identical populations of cells from the first cell-based assay with a vehicle or with no reagent; (d) comparing the results to determine the activity of the test chemical compound relative to the absence of the test chemical compound in the first cell-based assay;
(e) contacting the first of two populations of identical cells from the second cell-based assay with the test chemical compound; (f) contacting the second of two populations of identical cells from the second cell-based assay with the vehicle or with no reagent; (g) comparing the results to determine the activity of the test chemical compound relative to the absence of the test chemical compound in the second cell-based assay; and
(h) combining the results of steps to establish an activity profile for the test chemical compound in the assay panel; (3) assessing (M3) the potential safety/toxic or adverse effects/therapeutic or clinical efficacy or utility, of a chemical compound, comprising:
(a) using (m1) to establish an activity profile of a test chemical compound in an assay panel;
(b) using (m1) to establish an activity profile of a reference compound in the assay panel, the reference compound having established safety/toxic or adverse characteristics/therapeutic or clinical efficacy or utility;
(c) comparing the activity profile of the test chemical compound to the activity profile of the reference compound; and (d) if the activity profile of the test chemical compound is substantially similar to the activity profile of the reference compound, determining that the chemical compound has potential safety/toxic or adverse characteristics/therapeutic or clinical efficacy or utility, substantially similar to those of the reference compound; and (4) identifying cellular pathway(s) underlying drug toxicity, comprising:
(a) testing the effects of compound(s) with toxic or adverse effects against several proteins in intact cells; and (b) using the results of the tests to identify pathways associated with toxicity.
USE - For analyzing a test chemical compound (e.g. synthetic compound; combinatorial library element; natural product; peptide; antibody; recombinant or natural protein; known drug; pharmaceutical composition; toxicant; lead molecule; drug candidate; drug combination; agonist; antagonist; inhibitor; growth factor; hormone; vitamin; biological fluid or extract; cosmeceutical ingredient or product; nutraceutical ingredient or product; infectious agent,

or its component or antigen; poison, toxin, explosive or radioactive agent, or their product or component; and/or biological or chemical agent produced by a cell or organism in response to treatment with chemical, biological, infectious, poisonous, toxic or radioactive agent or their component) to identify an activity profile of the compound in cell(s); for assessing the potential safety/toxic or adverse effects/therapeutic or clinical efficacy or utility, of a chemical compound; for identifying cellular pathway(s) underlying drug toxicity. (All claimed.)

ADVANTAGE - The composition ascertains the mechanism of action of pharmacologically important compounds in the context of network biology, across the entire scope of the complex pathways of living cells; allows a rapid assessment of the on-pathway and off-pathway effects of lead compounds and drug candidates in living cells, and their comparisons with well-characterized drugs and toxicants to identify patterns with efficacy and toxicity; improves the drug discovery processes by identifying drug leads with desired safety and efficacy and in effecting early attrition of compounds with potential adverse effects in man; provides principles for pharmacological profiling of chemical compounds, drug candidates, established drugs and toxicants on a global scale; provides methods for assessing the activity, specificity, potency, time course, dose response and mechanism of action of chemical compounds in living cells; allows determination of the selectivity of a chemical compound within the biological context of any cell; allows detection of the potential off-pathway effects, adverse effects, or toxic effects of a chemical compound within the biological context of a particular cell type of interest; enables lead optimization, by performing pharmacological profiling of a collection or a series of lead compounds in an iterative manner until a desired pharmacological profile is obtained; enables attrition of drug candidates with undesirable or toxic properties; establishes pre-clinical safety profiles for new drug candidates; improves the efficiency of the drug discovery process by identifying unintended effects of lead compounds prior to clinical trials; improves the safety of first-in-class drugs by identifying adverse, toxic or other off-pathway effects prior to clinical trials; identifies positive or negative effects of drug excipients, carriers or drug delivery agents; provides methods suitable for the development of 'designer drugs' with predetermined properties; enables the identification of new therapeutic indications for known drugs; provides a method for analyzing the activity of any class of pharmacological agent on any biochemical pathway; enables the identification of the biochemical pathways underlying drug toxicity; enables the identification of the biochemical pathways underlying drug efficacy for a broad range of diseases; provides methods, assays and compositions useful for drug discovery and development; provides panels of assays suitable for pharmacological profiling; and is broadly applicable to any disease, pathway, gene, gene library, drug, drug target class, synthetic or natural product, chemical entity, assay format, detection mode, instrumentation, and cell type of interest.

L22 ANSWER 59 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2006-344255 [35] WPIDS
DOC. NO. CPI: C2006-113045 [35]
DOC. NO. NON-CPI: N2006-291734 [35]
TITLE: Method to identify glucose metabolic product comprises administering D7 glucose, allowing the glucose to form deuterated target metabolite, contacting the metabolite with mass tag to form tagged metabolite and detecting the mass
DERWENT CLASS: B04; D16; S03
INVENTOR: HALL M; HALL M P; SCHNEIDER L V; SCHNEIDER L
PATENT ASSIGNEE: (TARG-N) TARGET DISCOVERY INC

COUNTRY COUNT: 112

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2006050130	A2	20060511	(200635)*	EN	71[0]	
US 20060120961	A1	20060608	(200639)	EN		
EP 1824516	A2	20070829	(200757)	EN		
AU 2005302417	A1	20060511	(200763)	EN		
JP 2008519261	W	20080605	(200839)	JA	51	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2006050130	A2	WO 2005-US39017	20051028
US 20060120961	A1 Provisional	US 2004-623521P	20041029
AU 2005302417	A1	AU 2005-302417	20051028
EP 1824516	A2	EP 2005-824889	20051028
US 20060120961	A1	US 2005-262311	20051028
EP 1824516	A2	WO 2005-US39017	20051028
JP 2008519261	W	WO 2005-US39017	20051028
JP 2008519261	W	JP 2007-539190	20051028

FILING DETAILS:

PATENT NO	KIND		PATENT NO	
EP 1824516	A2	Based on	WO 2006050130	A
AU 2005302417	A1	Based on	WO 2006050130	A
JP 2008519261	W	Based on	WO 2006050130	A

PRIORITY APPLN. INFO: US 2004-623521P 20041029
US 2005-262311 20051028

AN 2006-344255 [35] WPIDS

AB WO 2006050130 A2 UPAB: 20060602

NOVELTY - Identifying a glucose metabolic product (I), comprising administering a D7 glucose (A), allowing (A) to be at least partially metabolized to form a deuterated target metabolite (B), separating (B) from the subject, contacting (B) with a mass tag and allowing the mass tag to attach to (B) to form a mass tagged deuterated target metabolite and detecting the mass of the mass tagged deuterated target metabolite to identify (I), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) analyzing metabolic pathways (II), comprising: administering a substrate (where the relative isotopic abundance of the isotope in the substrate is known), allowing the labeled substrate to be at least partially metabolized to form one or more target metabolites, and determining the abundance of the isotope in a plurality of target analytes in a sample from the subject to determine a value for the flux of each target analyte, where the plurality of target analytes comprise the substrate and/or one or more of the target metabolites; (2) screening for metabolites correlated with a disease or cellular state, comprising administering to a test subject and a control subject a substrate and the test subject has the disease, allowing the labeled substrate to be partially metabolized (where the conditions under which the administering and allowing steps are performed are the same for the test and control subject) and obtaining a sample from the test and control subject, determining for each sample the relative abundance of the isotope in a plurality of target analytes and comparing the values for flux for the test and control subjects a

difference in the flux value for a target analyte in the test subject and corresponding flux value for the control subject indicating that such analyte is potentially correlated with the disease; (3) screening for metabolites correlated with a disease comprises: analyzing a sample from a test subject having the disease and determining the isotopic abundance of the isotope in a plurality of analytes in the sample to determine a value for the flux of each analyte and comparing flux values for the analytes with flux values for the same analytes obtained for a control subject; (4) screening for the presence of a disease, comprising administering to a test subject a substrate, allowing sufficient time for the labeled substrate to metabolized, performing a plurality of electrophoretic methods in series to at least partially separate a plurality of target analytes from other biological components in a sample obtained from the test subject, determining a flux value for the target analytes, the flux value for each target analyte being determined from the abundance of the isotope in that analyte and comparing determined flux values with corresponding reference flux values for the same target analytes to assess the test subject's risk of disease; and (5) analyzing metabolites in an initial sample, comprising performing a plurality of capillary electrophoresis methods in series, each method comprising electrophoresing a sample containing multiple metabolites, to obtain a plurality of resolved metabolites (where the sample electrophoresed contains only a subset of the plurality of resolved metabolites from the immediately preceding method in the series, except the first method of the series in which the sample is the initial sample, the metabolites in the initial sample potentially containing one or more target analytes, the capillary electrophoresis methods) and analyzing fractions containing resolved metabolites from the final electrophoretic method to detect the presence of the target analytes.
USE - The method is useful forntifying a glucose metabolic product; and analyzing metabolic pathways. The method is useful forscreening for metabolites correlated with a disease (cancer, autism, microbial infection and digestive disorders) or cellular state (all claimed). The method is useful to detect a plurality of mass tagged deuterated metabolites; and determine the effect of chemical agents or combination of agents generally have no metabolism and the effect on the flux of certain metabolites of interest.
ADVANTAGE - The method reduce sample amount requirements and analysis time by eliminating multiple steps relative to the known method. The method provides determination of the sequence and the branching structure of a glycoform simulataneously.

L22 ANSWER 60 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2005-396144 [40] WPIDS
DOC. NO. CPI: C2005-122631 [40]
TITLE: Quantitating a population of target analytes in a sample, useful in biotechnology or pharmaceutical application, comprises using capture agents specific for immobilized small molecules and/or peptides/proteins
DERWENT CLASS: A96; B04; D16
INVENTOR: AFEYAN N B; GORDON N F; LEE F D; MENG X
PATENT ASSIGNEE: (AFEY-I) AFEYAN N B; (EPIT-N) EPITOME BIOSYSTEMS INC; (GORD-I) GORDON N F; (LEEF-I) LEE F D; (MENG-I) MENG X
COUNTRY COUNT: 106

PATENT INFO ABBR.:

PATENT NO	KIND DATE	WEEK	LA	PG	MAIN IPC
WO 2005050224	A2 20050602	(200540)*	EN	199[28]	
US 20050255491	A1 20051117	(200576)	EN		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2005050224 A2		WO 2004-US38539	20041115
US 20050255491 A1	Provisional	US 2003-519530P	20031113
US 20050255491 A1	Provisional	US 2003-532687P	20031224
US 20050255491 A1		US 2004-989226	20041115

PRIORITY APPLN. INFO: US 2003-532687P 20031224
US 2003-519530P 20031113
US 2004-989226 20041115

AN 2005-396144 [40] WPIDS

AB WO 2005050224 A2 UPAB: 20060203

NOVELTY - Quantitating (M1) a population of target analytes in a sample comprising using capture agents specific for immobilized small molecules and/or peptides/proteins, is new.

DETAILED DESCRIPTION - Quantitating (M1) a population of target analytes in a sample comprises:

(a) immobilizing the target analytes and/or their unique derivatives to a support, the unique derivatives, if used, predictably result from a treatment of the target analytes within the sample, where each of the target analytes or unique derivatives is immobilized on a series of distinct addressable locations on the support; (b) for each of the target analytes or unique derivatives, generating one or more capture agents that specifically bind the target analytes or derivatives;

(c) optionally, subjecting the sample to the treatment; (d) contacting the target analytes or unique derivatives on the support to a series of control samples, each within one of the series of distinct addressable locations, and each comprising a mixture of a fixed concentration of the capture agents and a variable concentration of the target analytes or their derivatives in solution; (e) generating a standard competition curve for each of the target analytes by measuring the amount of the capture agents bound to the target analytes or derivatives on the support; (f) contacting the target analytes or unique derivatives on the support to a mixture of the fixed concentration of the capture agent and the sample, in one of the series of distinct addressable locations, optionally after the treatment in step (c); and (g) determining the concentration of each of the target analytes, using each of the standard competition curves, by measuring the amount of the capture agent bound to the target analytes or derivatives on the support.

INDEPENDENT CLAIMS are also included for: (1) an array for detecting, profiling or quantitating target analytes in a sample, the array comprising a population of immobilized target analytes or their derivatives on a support, each of the target analytes is represented by at least one of the immobilized target analytes or their derivatives, the derivatives, if present, predictably result from a treatment of the sample, and each of the peptide fragments contains a PET unique to the fragments within the sample; (2) characterizing (M2) candidate antibodies for binding affinity; (3) an information database comprising a population of PET sequences, and optionally one or more nearest neighbors of each of the PET sequences; and property of antibodies specific for each of the PET sequences, the property including affinity towards the PET sequences, specificity towards the PET sequences against all other PET sequences and nearest neighbors, and performance of each of the antibodies in one or more in vitro or in vivo assays;

(4) designing arrays (M3) for large scale profiling of analyte levels for target analytes in a sample; (5) business methods for a biotechnology or pharmaceutical business;

(6) screening (M4) for marker(s) associated with a condition; and (7) an array of analytes manufactured or constructed by the above methods.

USE - The methods and arrays are used for detecting, profiling and quantitating a population of target analytes in a sample for use in diagnostic and/or research experimentation. The business method further comprises marketing and distributing the arrays for use in commercial and/or academic laboratories. (All claimed). The methods and arrays may be utilized in research and development in academic and industrial settings, medicine (predictive, preventive and personalized medicine, disease diagnosis - biomarker identification and measurement, etc.), pharmaceutical business (drug screening and development), natural and work environmental monitoring and protection, toxic substance control, and in food and cosmetic industry.

L22 ANSWER 61 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2004-080044 [08] WPIDS
CROSS REFERENCE: 2003-131580; 2003-596328
DOC. NO. CPI: C2004-032706 [08]
TITLE: Bioactive semi-purified extract useful in e.g. paints, inks comprises proteinaceous fluorescent dye obtained from female gonads of marine organism Holothuria scabra
DERWENT CLASS: B04; D21; E24
INVENTOR: GANGULY A; GOSWAMI U
PATENT ASSIGNEE: (COUL-C) COUNCIL SCI & IND RES; (COUL-C) COUNCIL SCI & IND RES SOUTH AFRICA; (COUL-C) CSIR COUNCIL SCI IND RES; (GANG-I) GANGULY A; (GOSW-I) GOSWAMI U; (COUN-N) COUNCIL SCI & IND RES INDIA
COUNTRY COUNT: 102

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC	
US 20020187202	A1	20021212	(200408)*	EN	71[22]		<--
							<--
WO 2003082989	A1	20031009	(200408)	EN			<--
							<--
AU 2003217441	A1	20031013	(200435)	EN			<--
EP 1490439	A1	20041229	(200502)	EN			
US 6916492	B2	20050712	(200546)	EN			
CN 1649968	A	20050803	(200578)	ZH			
JP 2006504808	W	20060209	(200612)	JA	69		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 20020187202	A1 CIP of	US 2001-820654	20010330
US 20020187202	A1	US 2002-107335	20020328
US 6916492	B2 CIP of	US 2001-820654	20010330
US 6916492	B2	US 2002-107335	20020328
AU 2003217441	A1	AU 2003-217441	20030324
CN 1649968	A	CN 2003-810116	20030324
EP 1490439	A1	EP 2003-712627	20030324
WO 2003082989	A1	WO 2003-IN70	20030324
EP 1490439	A1	WO 2003-IN70	20030324
JP 2006504808	W	JP 2003-580430	20030324
JP 2006504808	W	WO 2003-IN70	20030324

FILING DETAILS:

PATENT NO	KIND	PATENT NO

US 6916492 B2	CIP of	US 6582730 B
AU 2003217441 A1	Based on	WO 2003082989 A
EP 1490439 A1	Based on	WO 2003082989 A
JP 2006504808 W	Based on	WO 2003082989 A

PRIORITY APPLN. INFO: US 2002-107335 20020328
US 2001-820654 20010330

AN 2004-080044 [08] WPIDS

CR 2003-131580; 2003-596328

AB US 20020187202 A1 UPAB: 20060203

NOVELTY - A bioactive semi-purified extract comprises non-toxic proteinaceous fluorescent dye obtained from female gonads i.e. ovarian tissues of marine organism Holothuria scabra.

DETAILED DESCRIPTION - A bioactive semi-purified extract comprises non-toxic proteinaceous fluorescent dye obtained from female gonads i.e. ovarian tissues of marine organism Holothuria scabra (A), where fluorescent pigments of the dye give emissions in few to all wavelength ranges of UTVB, UVA, visible colored spectrums and infra- red spectrum. (A) is found in intertidal, submerged, shallow and deep waters, usually abundant in shaded areas such as alcoves, crevices, ledges, overhangings, rocky sandy habitats; is dull to bright colored with or without exo- and endo-skeleton, sessile, sedentary drifters, nektonic with varied swimming internal power; and is usually nocturnal in habit, liabile to active predation, with and without luminescent fluorescent pigments. Eggs of such organisms are fertilized externally. INDEPENDENT CLAIMS are included for the following: (1) fluorescent composition comprising the bioactive extract together with additives in the ratio of 1:20000 to obtain fluorescence of three colors at three different wavelengths; and (2) extraction of a natural fluorescent dye from Holothuria scabra sea-cucumber involving:

(a) collecting the material from seashore, changing of seawater and maintaining in the laboratory tanks without any mechanical aeration overnight; (b) dissecting the animals and removing the female gonads; (c) extracting the female gonads with 70% ethyl alcohol at least thrice without homogenization of tissues; and (d) obtaining the solution containing the fluorescent dye termed as Stock solution of the dye.

USE - As a natural fluorescent; in a fluorescent composition for preparation of coating compositions and inks; in detection of leaks; in undersea probes; as a fluorescent molecular probe in situ hybridization kits for molecular diagnostics; as a component of non-radioactive labeling and detection kits of biochemistry, cell biology, immunochemistry and molecular biology; in immuno-fluorescent detections; as a counterstain of DIG-labeled oligonucleotide probes and anti-DIG Fab-fragments; in single and multiple cell quantitative fluorescence in single and multicolor flowcytometry applications; for conducting experiments at field stations situated at subzero degree temperature area; as fluorochrome stains for epifluorescence microscopy; for a quick check of bio-contamination in the health food industry, cosmetic industry, pharmaceutical and chemical industries; for rapid estimations of bio-contaminants in laboratory cultures; for a rapid check of bio-pollutants under field conditions; as a competitive inhibitor of cholinesterases; in cell permeant dye compositions; as a fertility enhancer; to obtain a phase contrast and histochemical counterstain effect for different biochemical constituents of cells under transmitted light; in skin care composition; in number of paints, inks and textiles; for bleaching and brightening polymer; leak detection with a full spectrum fluorescent dye; in automated chemical metering system; to mark location of crashed air-crafts, life crafts, and equipment e.g. rockets; chromatophore sunscreen component of cosmetics creams and lotions; as fluorescent in situ hybridization application kit component for molecular diagnostics; as component of the non-radioactive labeling and detection kits of biochemistry, cell biology, immunochemistry, and molecular

biology for labeling of DNA, RNA, proteins and enzymes; for immunofluorescent detection; counterstain of DIG-labeled oligonucleotide probes and Anti-DIG Fab-fragments and single and multiple flow cytometry applications; fluorochrome stains for epifluorescence microscopy; for a quick check of biocontamination in the health food industry, cosmetic industry, pharmaceutical and chemical industries; for rapid estimations of biocontaminants in laboratory cultures; for a rapid check of biopollutants under field conditions; agglutination compositions; a natural colorant; a bioactive composition of the marine dye in the ratio of 1:20000 in ultrapure water to obtain fluorescences of three colors at six different wavelengths and a phase contrast effect under transmitted light; a dye for various fluorescent applications to be performed in areas of sub zero temperatures; for fertilization rate increase in aquaculture industry; for cell permeant membrane dye compositions; for identification of dead and live cells in tissue cultures; for dye compositions in bio-sensor; and as dye composition in molecular and microbiological kits (all claimed).

ADVANTAGE - The dye is stable at the room temperature and has a long shelf life. The molecular and radioactive kits of the dye can be exported at the room temperatures. The single dye has characteristics of at least 100 different fluorochromes e.g. DAPI, Hoechest 33258, Hoechest 33342, FITC, acridine orange, auramine, Rhodamine, TRITC, and propidium iodide, which are now in the market. The dye can be used in all applications where presently Plhycobiliproteins are used as unlike them the dye does not undergo loss in fluorescence upon freezing. The dye is a natural eco-friendly nontoxic cell permeant multiple fluorescent protein dye. Unlike most synthetic fluorescent dyes, the natural dye does not need to be mixed with another dye from getting different fluorescent hues at different wavelengths. The dye has agglutinating qualities. The dye enhances the rate of insemination of sperms and fertilization of eggs.

L22 ANSWER 62 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2003-103676 [09] WPIDS
DOC. NO. CPI: C2003-026334 [09]
DOC. NO. NON-CPI: N2003-082694 [09]
TITLE: Use of metallated porphyrin for treating or detecting diseases of cardiovascular system in combination with irradiation
DERWENT CLASS: A96; B02; P34; S03; S05
INVENTOR: GREENE S; LEITCH I M; ROBINSON B C; RYCHNOVSKY S
PATENT ASSIGNEE: (GREE-I) GREENE S; (LEIT-I) LEITCH I M; (MIRA-N) MIRAVANT PHARM INC; (ROBI-I) ROBINSON B C; (RYCH-I) RYCHNOVSKY S
COUNTRY COUNT: 99

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC	
WO 2002096366	A2	20021205	(200309)*	EN	247[0]		<--
							<--
US 20030105069	A1	20030605	(200339)	EN			<--
EP 1401506	A2	20040331	(200424)	EN			
							<--
AU 2002344234	A1	20021209	(200452)	EN			<--
JP 2004532251	W	20041021	(200469)	JA	379		
US 6827926	B2	20041207	(200480)	EN			
US 20050137180	A1	20050623	(200542)	EN			
AU 2002344234	B2	20071108	(200812)	EN			
AU 2008200847	A1	20080529	(200858)#	EN			

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002096366 A2		WO 2002-US17180	20020531
US 20030105069 A1	Provisional	US 2001-295345P	20010531
US 6827926 B2	Provisional	US 2001-295345P	20010531
US 20050137180 A1	Provisional	US 2001-295345P	20010531
AU 2002344234 A1		AU 2002-344234	20020531
AU 2002344234 B2		AU 2002-344234	20020531
EP 1401506 A2		EP 2002-744204	20020531
JP 2004532251 W		JP 2002-592879	20020531
US 20030105069 A1		US 2002-159005	20020531
US 6827926 B2		US 2002-159005	20020531
US 20050137180 A1	Div Ex	US 2002-159005	20020531
US 20050137180 A1		US 2004-965849	20041018
AU 2008200847 A1	Div Ex	AU 2002-344234	20020531
EP 1401506 A2	PCT Application	WO 2002-US17180	20020531
JP 2004532251 W	PCT Application	WO 2002-US17180	20020531
AU 2008200847 A1		AU 2008-200847	20080215

FILING DETAILS:

PATENT NO	KIND		PATENT NO	
US 20050137180	A1	Div ex	US 6827926	B
EP 1401506	A2	Based on	WO 2002096366	A
AU 2002344234	A1	Based on	WO 2002096366	A
JP 2004532251	W	Based on	WO 2002096366	A
AU 2002344234	B2	Based on	WO 2002096366	A

PRIORITY APPLN. INFO: US 2001-295345P 20010531
US 2002-159005 20020531
US 2004-965849 20041018
AU 2008-200847 20080215

AN 2003-103676 [09] WPIDS

AB WO 2002096366 A2 UPAB: 20060118

NOVELTY - Use of a metallated porphyrin (I) is claimed for treating or detecting diseases of the cardiovascular system by administering (I) that coordinates a metal in the central pyrrolic core and irradiating (I) with energy at a wavelength capable of exciting the molecule to give the desired detection or therapeutic effect.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following: (1) use of a tetrapyrrolic macrocycle (II) comprising a porphyrin, an azaporphyrin, a diazaporphyrin, a triazaporphyrin, a corrole, a porphycene, an isoporphycene, a hemiporphycene or a corrphycene, that coordinates gallium in the central pyrrolic core for treating or detecting diseases of the cardiovascular system; (2) restructuring the epithelial or endothelial layers of skin or stopping or arresting hair growth which comprises topically or systemically administering a tetrapyrrolic molecule that coordinates gallium in the central tetrapyrrolic core and irradiating the molecule with energy at a wavelength capable of exciting the molecule; (3) use of a gallium tetrapyrrole molecule for detecting or treating tissue which comprises locally, systemically, intramuscularly or intraperitoneally administering a gallium tetrapyrrolic molecule and irradiating the molecule with energy at a wavelength capable of exciting the molecule, where the tissue belongs to the hematological system, lymphatic system reticuloendothelial system, nervous system, endocrine and exocrine system, skeletomuscular system including bone, connective tissue, cartilage and skeletal muscle, pulmonary system, gastrointestinal system including the liver, reproductive system, immune

system, cardiovascular system, urinary system, auditory or olfactory system;
(4) treating diseases of the cardiovascular system which comprises
administering a tetrapyrrolic molecule that coordinates gallium in the central
tetrapyrrolic core and irradiating the graft with energy at a wavelength
capable of exciting the molecule, so that the graft is made less immunogenic
to the host, and
(5) new gallium porphyrin compounds e.g. compounds of formula (IA). R1, R2 =
CO2R3, CON(R4), CONHNH(R4), CON(R4)2, COR4, CON(R4)(R5), (CH2)nOH, (CH2)nOR6,
(CH2)n1CO2R7, (CHX)n1CO2R7, (CX2)n1CO2R7, (CH2)n1CONHR8, (CH2)n1CON(R8)2,
(CX2)n1CON(R8)R9, (CX2)n1CONHR8, (CX2)n1CON(R8)2, (CX2)n1CON(R8)R9,
(CH2)nNHR10, (CH2)nN(R10)2, (CH2)nN(R10)R11, S(R12), (CH2)nS(R13),
(CH2)nOPO2OR14, (CH2)nPO(OR14)2, (CH2)nPO2R14, (CH2)nPOR14, (CH2)nNHCOR15,
(CH2)nNHCOR15, (CH2)nNHNHCOR15, SO3R16, SO2NHR16, SO2N(R16)2, SO2N(R16)R17,
SO2NHNHR16 or SO2R16; R3, R7 = a counter ion or Q; Q = 1-20C alkyl or
heteroalkyl, aryl or heteroaryl, mono-, di- or poly-hydroxyalkyl, mono-, di-
or poly-hydroxyaryl or a functional group of less than 100000 daltons;
R4, R5 = Q, H, mono-, di- or poly-etheralkyl, mono-, di- or poly-etheraryl,
amino acid, amino acid ester or amino acid amide;
R6 = alkyl or heteroalkyl, aryl or heteroaryl, mono-, di- or poly-
hydroxyalkyl, mono-, di- or poly-hydroxyaryl or a functional group of less
than 100000 daltons;
n = 0-4;
n1 = 1-4
X = halo;
R8-R13 = Q, H, mono-, di- or poly-etheralkyl, or mono-, di- or poly-etheraryl;
R14 = H, Q, mono-, di- or poly-etheralkyl, mono-, di- or poly-etheraryl or a
counter ion;
R15 = 1-20C alkyl or heteroalkyl, aryl or heteroaryl or a functional group of
less than 100000 daltons; R16, R17 = H, a counter ion, 1-20C alkyl, haloalkyl,
heteroalkyl, haloheteroalkyl, aryl or heteroaryl, mono-, di- or poly-
hydroxyalkyl, mono-, di- or poly-etheralkyl, mono-, di- or poly-etheraryl,
amino acid, amino acid salt, amino acid ester, amino acid amide or a
functional group of less than 100000, and M = Ga3+ with an associated charge
balancing counter ion, provided that:
(1) R4 and R5 are not pentetic acid, polyfunctional carboxy compounds or
cyclen functional groups that bind metal ions with atomic numbers of 20-32,
37-39, 42-51 or 57-83; (2) R12 does not include carboxy, and (3) R1 and R2 are
not both COOH or both CO2Me. ACTIVITY - Cardiant; Antiarteriosclerotic;
Vasotropic; Vulnerary, Antipsoriatic; Immunosuppressive; Antiulcer;
Antiinflammatory; Fungicide; Dermatological; Antisebbhoreic; Cytostatic;
Virucide; Antibacterial.
Tests are described, but no relevant results are given in the source material.
MECHANISM OF ACTION - None given in the source material.
USE - Used for treating a vessel (e.g. an artery or a vein) wall or tissue
adjoining the vessel wall, or material attached to the vessel
wall of a coronary, carotid or peripheral vasculature, for treating or
detecting cardiovascular disease (e.g. atherosclerosis, restenosis and graft
disease), for treatment of restenosis (e.g. vessel wall negative geometric
remodeling, intimal thickening, increased intraluminal shear stress,
dysfunctional or absent endothelium, periadventitial fibrosis, increased motor
tone, fibrotic
contracture and scar formation) of occlusive tissue (e.g. foreign tissue, host
tissue, a tissue from an injury via invasive or non- invasive surgical
manipulation (such as suturing, vascular access, anastomosis, bypass
procedure, or shunt) formation induced in the vessel wall or by vascular
injury (e.g. via balloon angioplasty, stent deployment or injury from
endovascular device) to the vessel wall; for treatment of arteriovenous shunts
and for restructuring the
epithelia or endothelial layers of skin and for stopping or arresting
hair growth.

The methods are also used for the detection or treatment of tissue such as atherosclerotic plaque, for the treatment of disturbance of vascular and perivascular cellular processes selected from proliferation, replication, migration, necrosis, apoptosis, adhesion, matrix deposition, signaling pathways, paracrine and autocrine functions, mediator release, contraction, relaxation, shrinkage, phenotype changes, angiogenesis, aggregation, healing, repair, regulation of surrounding tissue, metabolism and matrices and in photodynamic therapy,
MRI diagnosis and radiodiagnostics.
ADVANTAGE - The compounds show good efficacy in advanced animal model systems and preferred uptake in the target tissue, with good clearance characteristics and low toxicity, have good uptake into cardiovascular tissues, show low myocardial tissue toxicity on light activation and are cleared rapidly from skin and other tissues.

L22 ANSWER 63 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 2000-293109 [25] WPIDS
DOC. NO. CPI: C2000-088632 [25]
TITLE: Isolated artemin growth factor proteins and the nucleic acids that encode them, useful for treating a range of degenerative neuronal disorders such as Parkinson's disease and Huntington's disease
DERWENT CLASS: B04; D16
INVENTOR: BALOH R H; MILBRANDT J D
PATENT ASSIGNEE: (BALO-I) BALOH R H; (MILB-I) MILBRANDT J D; (UNIW-C) UNIV WASHINGTON
COUNTRY COUNT: 88

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC	
WO 2000018799	A1	20000406	(200025)*	EN	94[15]		<--
AU 9964054	A	20000417	(200035)	EN			<--
EP 1028975	A1	20000823	(200041)	EN			<--
US 6284540	B1	20010904	(200154)	EN			<--
US 20020002269	A1	20020103	(200207)	EN			<--
JP 2002534957	W	20021022	(200301)	JA	117		<--
AU 764531	B	20030821	(200359)	EN			<--
NZ 509490	A	20031031	(200380)	EN			<--
MX 2001003244	A1	20030601	(200417)	ES			<--

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000018799	A1	WO 1999-US22604	19990929
US 6284540	B1 CIP of	US 1998-163283	19980929
US 20020002269	A1 CIP of	US 1998-163283	19980929
US 6284540	B1 Provisional	US 1998-108148P	19981112
US 20020002269	A1 Provisional	US 1998-108148P	19981112

US 6284540 B1 Div Ex	US 1998-218698 19981222
US 20020002269 A1 Div Ex	US 1998-218698 19981222
US 6284540 B1	US 1998-220528 19981224
US 20020002269 A1	US 1998-220920 19981224
AU 9964054 A	AU 1999-64054 19990929
AU 764531 B	AU 1999-64054 19990929
EP 1028975 A1	EP 1999-951657 19990929
NZ 509490 A	NZ 1999-509490 19990929
EP 1028975 A1	WO 1999-US22604 19990929
JP 2002534957 W	WO 1999-US22604 19990929
NZ 509490 A	WO 1999-US22604 19990929
MX 2001003244 A1	WO 1999-US22604 19990929
JP 2002534957 W	JP 2000-572257 19990929
MX 2001003244 A1	MX 2001-3244 20010328

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 764531 B	Previous Publ	AU 9964054 A
AU 9964054 A	Based on	WO 2000018799 A
EP 1028975 A1	Based on	WO 2000018799 A
JP 2002534957 W	Based on	WO 2000018799 A
AU 764531 B	Based on	WO 2000018799 A
NZ 509490 A	Based on	WO 2000018799 A
MX 2001003244 A1	Based on	WO 2000018799 A

PRIORITY APPLN. INFO: US 1998-218698 19981222
US 1998-163283 19980929
US 1998-108148P 19981112
US 1998-220528 19981224
US 1998-220920 19981224

AN 2000-293109 [25] WPIDS

AB WO 2000018799 A1 UPAB: 20050410

NOVELTY - Isolated artemin growth factor proteins and the nucleic acids that encode them, are new. Artemin is a neurotrophic factor that belongs to the GDNF (glial cell line-derived neurotrophic factor)/neurturin/persephin family of growth factors and promotes differentiation, maintains mature phenotype and provides trophic support, promoting growth and survival of neurons.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) an isolated and purified growth factor (I) comprising an artemin amino acid sequence (or conservatively substituted variant or fragment of at least 8 contiguous amino acids); (2) a pan-growth factor (II) comprising (I) and a fragment of at least 1 other growth factor from the TGF-beta (transforming growth factor-beta) superfamily;

(3) a nucleic acid encoding (II); (4) a composition (III) comprising (I) and a GFR-alpha (growth factor receptor-alpha) polypeptide; (5) an isolated polynucleotide (IV) comprising a sequence encoding (I) (and sequences (IV') complementary to (IV) or fragments of (IV) comprising at least 15 contiguous nucleotides); (6) a vector (V) comprising (IV) operably linked to expression regulatory elements;

(7) a host cell (VI) transformed with (V); (8) an isolated antibody (VI) that specifically reacts with (I); (9) a method (METH1) for detecting expression of an artemin polypeptide in a sample, comprising contacting the sample with (VI) and detecting binding of the antibody to the polypeptide; (10) a method (METH2) for detecting artemin mRNA in a sample, comprising detecting a polynucleotide in the sample that specifically hybridizes to a polynucleotide comprising (IVk) (a defined sequence given in the specification); and

(11) a method (METH3) for providing trophic support to and/or for producing differentiation of a cell, comprising treating the cell with the artemin (I) (or a fragment).
ACTIVITY - Antiparkinsonian; cerebroprotective; neuroprotective; antisclerotic; antialzheimers; nootropic; anticonvulsant; cytostatic; antimicrobial; gastrointestinal; laxative.
MECHANISM OF ACTION - (I) promotes the survival of trigeminal ganglion neurons, nodose ganglion neurons, superior cervical ganglion neurons and tyrosine-hydroxylase-expressing dopaminergic ventral midbrain neurons (claimed).
Artemin is the only member of the GDNF family that binds to GFR-alpha (growth factor receptor-alpha) and activates the GFR-alpha3/RET (Ret protein-tyrosine kinase) receptor complex and additionally, like GDNF and neurturin, artemin also binds to and activates GFRalpha1/RET. Embryonic day 14 ventral mesencephalon cultures were prepared by removing the entire mesencephalon from a rat into cold Leibovitz's L15 medium (containing 6 mg/ml glucose) and dissecting the tissue while keeping it on ice. Following dissection, the tissue was digested in a mixture of dispase (1 mg/ml) and collagenase (1 mg/ml) for 25 minutes (min). The tissue was then washed twice with modified N2 media and triturated 35 times. Cell density and viability was assessed using a hemocytometer to count trypan blue excluding cells. Cells were plated at 20000 cells/well on 8-well chamber slides (coated with 125 ng/ml poly-D-lysine and 25 ng/ml laminin) in serum-free medium consisting of DME (undefined)/Hams F12 (1:1), 1 mg/ml BSA (bovine serum albumin), 5 micromoles insulin, 10 nM progesterone, 100 micromoles putrescine, 30 nM selenium, 10 ng/ml rat transferrin, 100 U/ml penicillin, 100 U/ml streptomycin. Factors were added within 15 min of plating. After 3 days in culture, the cells were fixed, stained for tyrosine hydroxylase, and the number of tyrosine hydroxylase staining (TH+) neurons were counted. The results from two independent experiments are determined as the percentage of surviving TH+ neurons over control. Interestingly, artemin supported the survival of dopaminergic midbrain neurons, although there was no apparent artemin expression in the ventral midbrain. Therefore, artemin was found to promote survival of central as well as peripheral neurons, and this indicates that receptors for artemin are present in both these populations. In summary, all of the biological responses of artemin in peripheral and central neurons, and in neuronal cell lines, suggested that artemin utilizes receptor components similar to or overlapping with GDNF and neurturin.
USE - The polypeptides, or nucleic acids encoding them, are administered to treat peripheral neuropathy, amylotrophic lateral sclerosis, Alzheimer's disease, Parkinson's disease, Huntington's disease, ischemic stroke, acute brain injury, acute spinal cord injury, a nervous system tumor (e.g. blastomas), multiple sclerosis, infection or enteric disease (e.g. idiopathic constipation or constipation associated with Parkinson's disease, spinal cord injury or use of opiate pain killers). They may also be used to treat a patient suffering from small cell lung carcinoma. The polypeptides or nucleic acids may be used in this way to rectify mutations in a patient's genome that result in decreased protein expression or expression of inactive polypeptides. The proteins may also be used to identify modulators (agonists, antagonists and/or antibodies) of artemin expression and activity. The antibodies may then be used to detect and quantify the presence of artemin proteins in a sample and to down regulate artemin activity. The nucleic acids may also be used to detect corresponding nucleic acids in sample (e.g. by polymerase chain reaction (PCR) analysis) (claimed).

L22 ANSWER 64 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 1998-343278 [30] WPIDS
DOC. NO. CPI: C1998-105839 [30]
TITLE: Antitumour substance NSCL-96F037 is a cell cycle

inhibitor - useful as active ingredient in antitumour agents, obtainable from Aspergillus ustus
DERWENT CLASS: B04; D16
INVENTOR: ASARI T; FUKUMOTO K; HARADA T; KANO K; KAWASHIMA H; KONO S; OMIZO K; SEKIYA H
PATENT ASSIGNEE: (YAWH-C) NIPPON STEEL CHEM CO; (YAWA-C) NIPPON STEEL CORP
COUNTRY COUNT: 1

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC	
JP 10130266	A	19980519	(199830)*	JA	14[8]		<-- <--
JP 3131574	B2	20010205	(200110)	JA	14		<--

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
JP 10130266 A		JP 1997-188749	19970714
JP 3131574 B2		JP 1997-188749	19970714

FILING DETAILS:

PATENT NO	KIND	PATENT NO
JP 3131574 B2	Previous Publ	JP 10130266 A

PRIORITY APPLN. INFO: JP 1996-234477 19960904
AN 1998-343278 [30] WPIDS
AB JP 10130266 A UPAB: 20050521
Antitumour substance NSCL-96F037 (I) has the following properties: (i) molecular weight: 350 (FABMS M/Z 351 (M+H)); (ii) molecular formula: C20H22N4O2; (iii) IR spectrum: as shown in figure 1; (iv) 1H-NMR spectrum (500 MHz, in CDCl3, internal standard: chemical shift value of CHCl3 is defined to be 7.24 ppm): δ (ppm) as indicated in specification; (v) 13C-NMR spectrum (400 MHz, in CDCl3, internal standard: chemical shift value of CDCl3 is defined to be 77.10 ppm): δ (ppm): as indicated in specification; (vi) 15N-NMR spectrum (600MHz, in CDCl3, internal standard: chemical shift value of ammonia is defined to be 0 ppm): δ (ppm) 112, 134, 161 and 253; (vii) UV absorption spectrum in methanol has a maximum value at 323 nm under neutral condition and has a peak at 230 nm; (viii) is excited by UV ray of 320-340 nm in methanol under neutral condition and has a maximum value at 395-400 nm and emits fluorescence having a wave length range of 350 -550 nm; (ix) is soluble in ethyl acetate, chloroform, methanol and pyridine; hardly soluble in water, benzene and toluol; (x) negative to ninhydrin reaction; positive to colour reaction with nitrite ion (orange); and (xi) colour: white. Also claimed is Aspergillus ustus having the ability to produce (I).
MORE SPECIFICALLY - (I) is of formula (I).
USE - (I) is a cell cycle inhibitor and is used in antitumour agents as the active ingredient (claimed).
ADVANTAGE - (I) has a high animal cell specific growth inhibiting activity and cell cycle inhibiting activity.

Member(0002)
ABEQ JP 3131574 B2 UPAB 20050521
Antitumour substance NSCL-96F037 (I) has the following properties: (i)

molecular weight: 350 (FABMS M/Z 351 (M+H)); (ii) molecular formula: C20H22N4O2; (iii) IR spectrum: as shown in figure 1; (iv) 1H-NMR spectrum (500 MHz, in CDCl3, internal standard: chemical shift value of CHCl3 is defined to be 7.24 ppm): δ (ppm) as indicated in specification; (v) 13C-NMR spectrum (400 MHz, in CDCl3, internal standard: chemical shift value of CDCl3 is defined to be 77.10 ppm): δ (ppm): as indicated in specification; (vi) 15N-NMR spectrum (600MHz, in CDCl3, internal standard: chemical shift value of ammonia is defined to be 0 ppm): δ (ppm) 112, 134, 161 and 253; (vii) UV absorption spectrum in methanol has a maximum value at 323 nm under neutral condition and has a peak at 230 nm; (viii) is excited by UV ray of 320-340 nm in methanol under neutral condition and has a maximum value at 395-400 nm and emits fluorescence having a wave length range of 350 -550 nm; (ix) is soluble in ethyl acetate, chloroform, methanol and pyridine; hardly soluble in water, benzene and toluol; (x) negative to ninhydrin reaction; positive to colour reaction with nitrite ion (orange); and (xi) colour: white. Also claimed is Aspergillus ustus having the ability to produce (I).

MORE SPECIFICALLY - (I) is of formula (I).

USE - (I) is a cell cycle inhibitor and is used in antitumour agents as the active ingredient (claimed).

ADVANTAGE - (I) has a high animal cell specific growth inhibiting activity and cell cycle inhibiting activity.

L22 ANSWER 65 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 1997-387441 [36] WPIDS
CROSS REFERENCE: 1997-431465
DOC. NO. CPI: C1997-124408 [36]
TITLE: New bioactive substances - suppress growth of interleukin-6-dependent MH-60 cells, used in treatment of diseases involving interleukin-6, cancer cachexia, multiple myeloma or rheumatoid arthritis
DERWENT CLASS: B02; D16
INVENTOR: HAYASHI M; KOMIYAMA K; OMURA S; TAKAMATSU S
PATENT ASSIGNEE: (KITA-C) KITASATO INST
COUNTRY COUNT: 5

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC	
EP 787733	A2	19970806	(199736)*	EN	23[8]		<--
CA 2195291	A	19970720	(199748)	EN			<--
US 5756320	A	19980526	(199828)	EN			<--
EP 787733	B1	20011205	(200203)	EN			<--
DE 69708712	E	20020117	(200213)	DE			<--

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 787733 A2		EP 1997-300277	19970117
CA 2195291 A		CA 1997-2195291	19970116
DE 69708712 E		DE 1997-69708712	19970117

DE 69708712 E EP 1997-300277 19970117
US 5756320 A US 1997-785767 19970121

FILING DETAILS:

PATENT NO	KIND	PATENT NO
DE 69708712 E	Based on	EP 787733 A

PRIORITY APPLN. INFO: JP 1996-7521 19960119
AN 1997-387441 [36] WPIDS
CR 1997-431465
AB EP 787733 A2 UPAB: 20050518
New stereoisomers of formula (I) are new:
USE - (I), K93-0711 I-1 and K93-0711 I-2 are used in the treatment of diseases involving interleukin (IL)-6, cancer cachexia, multiple myeloma or rheumatoid arthritis (all claimed). The compounds suppress growth of IL-6-dependent MH-60 cells.

Member(0003)
ABEQ US 5756320 A UPAB 20050518
New stereoisomers of formula (I) are new:
USE - (I), K93-0711 I-1 and K93-0711 I-2 are used in the treatment of diseases involving interleukin (IL)-6, cancer cachexia, multiple myeloma or rheumatoid arthritis (all claimed). The compounds suppress growth of IL-6-dependent MH-60 cells.

L22 ANSWER 66 OF 66 WPIDS COPYRIGHT 2009 THOMSON REUTERS on STN
ACCESSION NUMBER: 1995-320408 [41] WPIDS
DOC. NO. CPI: C1995-142322 [41]
TITLE: Immuno-modulating compsn. containing extract Buxacea leaves - is a stimulatory on quiescent cells and a suppressant on activated cells partic. for treating HIV infections or in vitro stimulation of lymphocytes
DERWENT CLASS: B04
INVENTOR: CONDOM R; DELLAMONICA P; DURANT J; GUEDJ R; PATINO N; REBOUILLAT A
PATENT ASSIGNEE: (DELL-I) DELLAMONICA P; (DURA-I) DURANT J; (REBO-I) REBOUILLAT A; (UYNI-N) UNIV NICE-SOPHIA ANTIPOLIS
COUNTRY COUNT: 19

PATENT INFO ABBR.:

PATENT NO	KIND DATE	WEEK	LA	PG	MAIN IPC	
WO 9523606	A1 19950908	(199541)*	FR	32[7]		<--
FR 2716805	A1 19950908	(199541)	FR	28[6]		<--
EP 804213	A1 19971105	(199749)	FR			<--

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9523606 A1		WO 1995-FR247	19950302
FR 2716805 A1		FR 1994-2514	19940304
EP 804213 A1		EP 1995-911368	19950302
EP 804213 A1		WO 1995-FR247	19950302

FILING DETAILS:

PATENT NO	KIND	PATENT NO
EP 804213 A1	Based on	WO 9523606 A

PRIORITY APPLN. INFO: FR 1994-2514 19940304
AN 1995-320408 [41] WPIDS
AB WO 1995023606 A1 UPAB: 20050702
Immunomodulatory compsn. (A) contains active ingredients of a plant extract that has been prepared by: (1) extracting leaves of a plant of the family Buxacea with water-organic solvent mixture then removing solvent; (2) suspending the crude extract in aqueous alcoholic solution then applying to a nonionic, macroporous resin which is eluted with aqueous alcohol to recover a sugar-free extract; (3) removing elution solvent and dissolving the residue in water; (4) applying the solution to a weakly acidic cation exchange resin and eluting with water; (5) removing solvent from the eluate and dissolving the residual solid in aqueous solution of acid pH; and (6) applying the solution to a strongly acidic cation exchange resin and eluting with water to recover an alkaloid-free extract.
USE - (A) stimulates basal production of IL-1beta, TNFalpha and GM-CSF by peripheral blood mononuclear cells, but inhibits stimulation of these cpds. by other activators and does not stimulate IL-2 or IL-6. It reinforces the inhibitory effect of cyclosporin on IL-2 but antagonises inhibition of cell proliferation induced by cyclosporin. Generally (A) is immunostimulatory on quiescent cells but immunosuppressant on activated cells. Partic. it is used to treat viral infections especially those caused by HIV, but may also be used (not claimed) in organ transplants. (A) can also be used for in vitro stimulation of lymphocytes.
ADVANTAGE - (A) is well tolerated by HIV-infected subject and causes an increase in the number of CD4 positive cells.

Member(0001)
ABEQ FR 2716805 A1 UPAB 20050702
Immunomodulatory compsn. (A) contains active ingredients of a plant extract that has been prepd. by: (1) extracting leaves of a plant of the family Buxacea with water-organic solvent mixt. then removing solvent; (2) suspending the crude extract in aq. alcoholic soln. then applying to a nonionic, macroporous resin which is eluted with aq. alcohol to recover a sugar-free extract; (3) removing elution solvent and dissolving the residue in water; (4) applying the soln. to a weakly acidic cation exchange resin and eluting with water; (5) removing solvent from the eluate and dissolving the residual solid in aq. soln. of acid pH; and (6) applying the soln. to a strongly acidic cation exchange resin and eluting with water to recover an alkaloid-free extract.
USE - (A) stimulates basal prodn. of IL-1beta, TNFalpha and GM-CSF by peripheral blood mononuclear cells, but inhibits stimulation of these cpds. by other activators and does not stimulate IL-2 or IL-6. It reinforces the inhibitory effect of cyclosporin on IL-2 but antagonises inhibition of cell proliferation induced by cyclosporin. Generally (A) is immunostimulatory on quiescent cells but immunosuppressant on activated cells. Partic. it is used to treat viral infections esp. those caused by HIV, but may also be used (not claimed) in organ transplants. (A) can also be used for in vitro stimulation of lymphocytes.
ADVANTAGE - (A) is well tolerated by HIV-infected subject and causes an increase in the number of CD4 positive cells.

SEARCH HISTORY

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=> d his ful

     (FILE 'HOME' ENTERED AT 10:11:14 ON 30 DEC 2009)

     FILE 'HCAPLUS' ENTERED AT 10:13:06 ON 30 DEC 2009
                E CHAIKEN JOSEPH/AU
L1            27 SEA ABB=ON  ("CHAIKEN JOE"/AU OR "CHAIKEN JOHN"/AU OR "CHAIKEN
                   JOSEPH"/AU)
                E DRACKER ROBERT/AU
L2             3 SEA ABB=ON  ("DRACKER ROBERT"/AU OR "DRACKER ROBERT A"/AU)
                E HAGRMAN PAMELA J/AU
L3            19 SEA ABB=ON  ("HAGRMAN P J"/AU OR "HAGRMAN PAMELA"/AU OR
                   "HAGRMAN PAMELA J"/AU OR "HAGRMAN PAMELA JAYNE"/AU)
                E HAGRMAN DOUGLAS/AU
L4            28 SEA ABB=ON  ("HAGRMAN DOUGLAS"/AU OR "HAGRMAN DOUGLAS E"/AU OR
                   "HAGRMAN DOUGLAS EARL"/AU)
L5             1 SEA ABB=ON  L1 AND L2 AND L3 AND L4
L6        822558 SEA ABB=ON  (?CELL? OR ?MEMBRAN?)(5A)(?VIABIL? OR ?LIVING? OR
                   ?LIVE? OR ?PROLIF? OR ?MULT? OR ?GROW? OR ?DUPLICAT? OR
                   ?METABOL?)
L7          8372 SEA ABB=ON  L6 AND (?RAMAN? OR ?INFRARED? OR ?INFRA?(W)RED? OR
                   ?NEAR?(W)(?INFRARED? OR ?SPECTROSC? OR ?SPECTROSC?))
L8            32 SEA ABB=ON  L7 AND (?DEUTERAT? OR ?DEUT? OR ?TRIT?)(L)(?MATERIA
                   L? OR ?METABOL? OR ?GLUCOS? OR ?DEXTROS? OR ?SUBSTITUENT?)
L9          8372 SEA ABB=ON  L7 OR L8
L10            5 SEA ABB=ON  L9 AND (?TRITIAT?(4A)(?THIAMEDENE? OR ?THYMIDINE?))
L11         8372 SEA ABB=ON  L9 OR L10
L12         2723 SEA ABB=ON  L11 AND (?BACTER? OR ?MICROB? OR ?FUNG? OR
                   ?PROTOZ? OR D20 OR ACID? OR ?WATER? OR ?DEUTERIUM?)
L13         2723 SEA ABB=ON  L12 AND (?RAMAN? OR ?INFRARED? OR ?INFRA?(W)RED?
                   OR ?NEAR?(W)(?INFRARED? OR ?SPECTROSC? OR ?SPECTROSC?))
L14           22 SEA ABB=ON  L13 AND (?DEUTERAT? OR ?DEUT? OR ?TRIT?)(L)(?MATERI
                   AL? OR ?METABOL? OR ?GLUCOS? OR ?DEXTROS? OR ?SUBSTITUENT?)
L15            4 SEA ABB=ON  L14 AND ?INTEG?
L16           22 SEA ABB=ON  L14 OR L15

     FILE 'MEDLINE, BIOSIS, EMBASE, DRUGU' ENTERED AT 10:23:06 ON 30 DEC 2009
L17           34 SEA ABB=ON  L16

     FILE 'HCAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 10:28:08 ON 30 DEC 2009
L18           40 DUP REMOV L16 L17 (16 DUPLICATES REMOVED)

     FILE 'WPIDS' ENTERED AT 10:28:18 ON 30 DEC 2009
L19           27 SEA ABB=ON  L14 OR L15

     FILE 'HCAPLUS, MEDLINE, BIOSIS, EMBASE, WPIDS' ENTERED AT 10:31:36 ON 30
     DEC 2009
L20           66 DUP REMOV L18 L19 (1 DUPLICATE REMOVED)
L21           28 SEA ABB=ON  L20 AND (PRD<20031222 OR PD<20031222)
L22           66 SEA ABB=ON  L20 OR L21

     FILE HOME

     FILE HCAPLUS

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FILE COVERS 1907 - 30 Dec 2009 VOL 152 ISS 1
FILE LAST UPDATED: 29 Dec 2009 (20091229/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Oct 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Oct 2009

HCAplus now includes complete International Patent Classification (IPC) reclassification data for the third quarter of 2009.

CAS Information Use Policies apply and are available at:

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This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE MEDLINE
FILE LAST UPDATED: 29 Dec 2009 (20091229/UP). FILE COVERS 1949 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2010 Medical Subject Headings (MeSH) vocabulary and tree numbers from the U.S. National Libra of Medicine (NLM). Additional information is available at

http://www.nlm.nih.gov/pubs/techbull/nd09/nd09_medline_data_changes_2010.

See HELP RLOAD for details.

MEDLINE was last reloaded on February 21, 2009.

This file contains CAS Registry Numbers for easy and accurate substance identification.

See HELP RANGE before carrying out any RANGE search.

FILE BIOSIS
FILE COVERS 1926 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1926 TO DATE.

RECORDS LAST ADDED: 23 December 2009 (20091223/ED)

BIOSIS has been augmented with 1.8 million archival records from 1926 through 1968. These records have been re-indexed to match current BIOSIS indexing.

FILE EMBASE
FILE COVERS 1974 TO 29 Dec 2009 (20091229/ED)

EMBASE was reloaded on March 30, 2008.

EMBASE is now updated daily. SDI frequency remains weekly (default) and biweekly.

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Beginning January 2008, Elsevier will no longer provide EMTREE codes as part of the EMTREE thesaurus in EMBASE. Please update your current-awareness alerts (SDIs) if they contain EMTREE codes.

For further assistance, please contact your local helpdesk.

FILE DRUGU
FILE LAST UPDATED: 28 DEC 2009 <20091228/UP>
>>> DERWENT DRUG FILE (SUBSCRIBER) <<<

>>> FILE COVERS 1983 TO DATE <<<
>>> THESAURUS AVAILABLE IN /CT <<<

FILE WPIDS
FILE LAST UPDATED: 22 DEC 2009 <20091222/UP>
MOST RECENT UPDATE: 200982 <200982/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> Now containing more than 1.5 million chemical structures in DCR <<<

>>> IPC, ECLA, US National Classifications and Japanese F-Terms and FI-Terms have been updated with reclassifications to end of September 2009.
No update date (UP) has been created for the reclassified documents, but they can be identified by specific update codes (see HELP CLA for details) <<<

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FOR DETAILS OF THE PATENTS COVERED IN CURRENT UPDATES, SEE
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>>> HELP for European Patent Classifications see HELP ECLA, HELP ICO <<<

>>> Japanese FI-TERM thesaurus in field /FCL added --> see NEWS <<<